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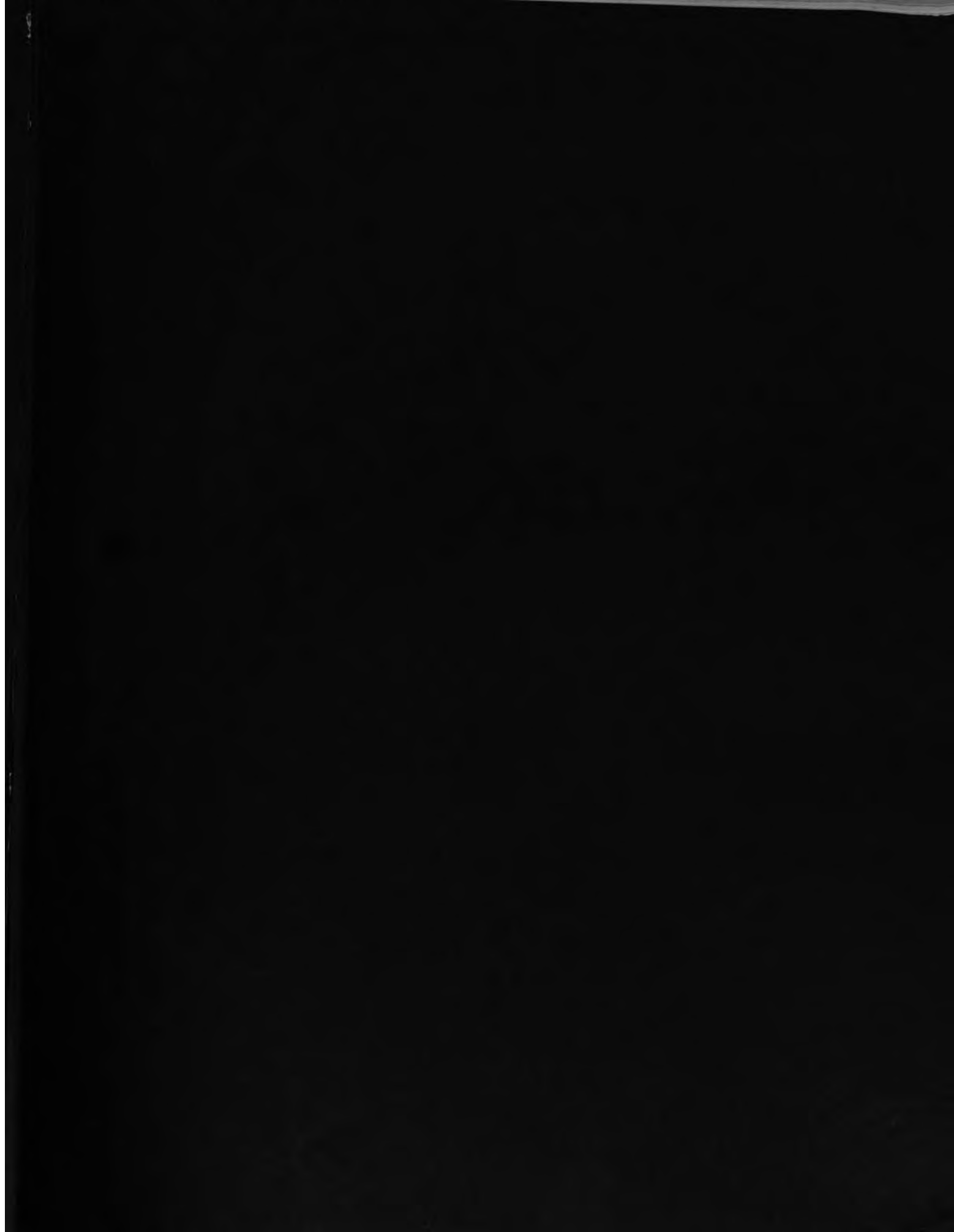


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THE
Journal of Tropical Medicine

*A MONTHLY JOURNAL DEVOTED TO MEDICAL, SURGICAL AND
GYNÆCOLOGICAL WORK IN THE TROPICS*

EDITED BY
JAMES CANTLIE, M.B., F.R.C.S., AND W. J. SIMPSON, M.D., F.R.C.P.

VOLUME II.
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THE ETIOLOGY OF MALARIAL FEVER.¹

By GEORGE THIN, M.D.,
President of the Section.

THE programme which has been arranged for our Section is ample enough to have justified the suppression of the usual introductory address; and if I had consulted my own wishes I should have invited you to begin without delay the important work that is before us. But there are exceptional circumstances which lead me to think that some introductory remarks may be useful.

THE MOSQUITO THEORY.

At the last meeting of this Association, when the Section for Tropical Diseases was constituted, your distinguished first President was privileged to make to you the announcement of a discovery of the first importance. Major Ross, after several years' laborious work, guided by scientific instincts of a high order, and characterised by a tenacity and strength worthy to be put in comparison with the best feats—and that is not saying little—of other eminent Scotchmen whose names are indissolubly bound up with the history of our Indian Empire, succeeded in finding the key to a problem which had long puzzled the ablest and most careful students of the etiology of malarial fevers. The coincidence of the fever season

in malarial countries with the period of activity of mosquito life, the danger of exposure to night air, night being the time when mosquitos most actively bite, had long struck observers. I find, from an admirable summary by Dr. Nuttall, published in the twenty-fifth volume of the *Centralblatt für Bakteriologie*, that the causation of malarial fever by insects was referred to by Nott in 1848 at New Orleans as something already known, whilst in 1883 Dr. King published in America an excellent exposition of the mosquito theory. This theory was again suggested by Laveran in 1891, and also by Flüge in the same year. In 1892 Pfeiffer, in a paper on the "Coccidium of the Rabbit," in which he showed that this parasite is capable of two different cycles of development, one being exogenous, mentions that Koch had suggested that a similar condition might hold good for the parasite of malaria, and that exogenous malarial spores might be conveyed to man through the agency of blood-sucking insects such as mosquitos.

THE WORK OF MANSON AND ROSS.

In 1894 Dr. Manson, in his Goulstonian Lectures, brought this problem prominently before medical men in this country, and urged that a mosquito theory was best calculated to explain the various conditions of the problem. His suggestion was that, similarly to what takes place in the case of filaria Bancrofti, in connection with which his own work is so well known and appreciated, the female mosquito fills herself with infected blood, lays her eggs some days later, and dies beside them. The water then becomes contaminated with the spores of the parasite of malaria, through which they might be taken into the human body, or they might be breathed in the dust of dried-up pools, or the larvæ coming out of the eggs might

¹ An address delivered at the opening of the Section of Tropical Diseases, at the Annual Meeting of the British Medical Association at Portsmouth, August, 1899. Printed by permission of the *British Medical Journal*.

eat them with the body of the mother; or the ground might become infected by mosquitos containing the parasite falling and dying on it. I take this brief summary from Nuttall's paper. (Dr. Manson, who is happily with us, will, I hope, in one of our discussions, give us his latest views on this important subject.) King, Laveran, Bignami, Mendini, and Koch, on the other hand, considered that the infection is conveyed by the direct bite of the mosquito.

So far we have been dealing with theories, more or less suggested and justified by reasoning from recognised facts, but no direct proof had been forthcoming: whilst, on the other hand, there were conditions which seemed to render a mosquito theory improbable. It was well known that mosquitos might prevail in abundance in localities in which no malarial fever occurred. It was pointed out to me, for example, during a visit I paid to Sicily a few years ago, that a very narrow line separated the fever from the fever-free zone, whilst mosquitos prevailed equally in both, and some distinguished physicians, of great experience in the climate and in malaria, to whom I spoke of Ross's earlier work and the importance it seemed to me to have, found these objections insuperable. Ross's discoveries at once and for ever removed these objections. From the time that he showed that to inoculate sparrows with proteosoma by the medium of the mosquito a special mosquito was necessary, it was evident that the next thing to do was to discover whether some particular genus or species of mosquito was the host of the parasite of malaria. Already in August, 1897, Ross when examining two "dappled-winged" mosquitos which had been fed on a patient who had crescents in his blood, had found in the middle intestine certain round bodies which contained malarial pigment and which he rightly regarded as a development of the parasite.

I had hoped that we should have had the gratification of hearing from Major Ross's own lips the account of the interesting story of how, inspired and encouraged by Dr. Manson's advice and experience, he undertook the solution of the malarial problem, and finally succeeded in removing what had previously been only theory to the domain of ascertained fact; but, as you know, he has left on an expedition to the West Coast of Africa to continue his researches, and I need hardly say with what expectation we look forward to the results of his work in that fever-stricken land.

THE WORK OF THE ITALIAN INVESTIGATORS.

Major Ross's investigations in India have been too recently described in the pages of the *British Medical Journal* to make it necessary for me to refer to them further. I shall now, therefore, proceed to give a brief exposition of what has been done during the past year by Grassi, Bignami, and Bastianelli in Rome, the result of whose labours has been to throw much light on many of the conditions that regulate the propagation of human malaria by the agency of mosquitos.

Whilst Ross was working in Calcutta with proteosoma, the probable connection between mosquitos and malaria was being studied from another side by the distinguished Italian naturalist Grassi. During the summer of 1898 he made an exhaustive study of

the mosquitos that prevail in different parts of Italy, and he found that there were many places where *Culex pipiens*, the predominating mosquito, was common, but in which there was no indigenous malaria; whilst in places where malaria occurs he found a large mosquito, called by zoologists *Anopheles claviger*. This species, characterised by four spots on the wings arranged like a capital T, may, he says, be defined as the true talebearer of the presence of malaria. He has mentioned a number of malarial places in Italy in which this holds good, and he refers to malarial zones existing in the midst of non-malarial districts. In these zones he found *Anopheles claviger*, but in the surrounding free zones the mosquitos were of other genera. At one part where he found tertian ague very prevalent he found this mosquito enormously diffused, and he associated it with the fever. He finally came to the conclusion that in connection with malaria in Italy all mosquitos may be excluded except three species, the characteristic marks and habits of which he has described at length. *Anopheles claviger*, to which fever could be most clearly traced, bites sometimes during the day and night, but mostly half an hour before and half an hour after sunset.

THE EXPERIMENTAL PROOF.

Such was the condition of our knowledge of this subject when Bignami first succeeded in getting positive results which supported his theory of the direct inoculation of malaria by mosquito bites. His first experiments, in which a number of adult mosquitos were brought from a malarial station and set free in a suitable room in Rome, were made in 1894. The result was negative. Another series of experiments was begun early in 1898, and again had a negative result. A second experiment in that year, which was begun on August 24, also had a negative result; but at the end of September Grassi's observations showing that malaria in Italy was associated with the prevalence of particular mosquitos, and Ross's papers showing that a special mosquito was needed as the host of the proteosoma of the sparrow, were published, and at once suggested to Bignami the course which he had to take. The same man who had voluntarily submitted himself to the experiment in August, and in which the results were negative, again submitted himself to experiment. Bignami gives an account of the history of this man, which shows that he had never been exposed to the chances of malarial infection. Guided by Grassi's information, mosquitos were first brought on October 10 from Maccarese, where fever prevailed, and were identified by Grassi as *Culex penicillaris*, *Culex malariae*, and *Anopheles*. The experiment begun on September 26 was suspended on October 3 for want of mosquitos, and was resumed on the 10th to the 14th with new supplies. On the 23rd a new but scanty supply was again got. During the last days of October the subject of the experiment began to suffer from symptoms of fever, which developed in an unmistakable manner on November 1, and was cured on November 3 by hypodermic injections of quinine. By November 7 the patient was perfectly free from fever, and rapidly regained his appetite and strength. On November 2 the blood carefully examined gave a

negative result. On the 3rd a few young ring-shaped parasites, moving or discoid, without pigment, with characteristic appearances of the summer-autumn parasites, were found, increasing in the course of the day and being very numerous in the afternoon, when very fine granules of pigment were found on the edge. Dried and stained preparations showed the characteristic appearances of the parasite. In the district in Rome in which the experiment was carried out there had not been a case of spontaneous fever within the memory of medical men, the centre of Rome being absolutely free from fever. The type of fever was that which prevailed at Maccaresse, from whence the mosquitos were brought.

This result has since been amply confirmed by further experiments by Bignami and Bastianelli, and it has now been conclusively shown that anopheles may be made to bite a man suffering from malarial fever, and if then kept at a proper temperature (30° C.) for the requisite number of days to allow the sporozoites to be present in the salivary glands, and then made to bite a healthy man who is free from malarial fever, this man will, after the regular incubation period, develop malaria; and it is important to note that the parasite so conveyed from man to man through the mosquito retains its specific type. If the source of supply gives in the first instance crescents, the mosquito conveys to the healthy man the corresponding fever of the summer-autumn type. They have also shown by microscopical examination that in the different stages of development in the body of the mosquito the crescents and tertian parasites retain their distinguishing characteristics. It further results from their experiments that a very few bites are sufficient to convey fever; probably a single one sufficing. What is important is the species of mosquito and the stage of development of the parasite in its body. They infer that in the course of the winter the parasites in anopheles die out, and that infection is conveyed in the spring by the insects biting persons who suffer from relapses of fever from the previous season, and then biting healthy persons after the sporozoites have developed.

THE PARASITE AND THE HOST.

Grassi, Bignami and Bastianelli have made a series of observations and experiments on the development of the parasite in the body of anopheles, and have collected much valuable information regarding the habits and life history of that insect. The development of the parasite of malaria in the body of anopheles seems to be identical with the development of the proteosoma in the "grey mosquito" of Ross. If the mosquitos are placed in a temperature of 14° or 15° C. after they bite, no development takes place. Development takes place at 20° to 22° C., but more slowly than at 30° C. The observations which they record refer to cultivations at the latter temperature. They have found that in order that crescents should develop in anopheles it is necessary that they should be ripe. For the first 40 hours the crescents are still mostly fusiform in shape, being almost identical with those observed in fresh preparations, except that they are larger and the pigment is differently disposed. In preparations stained at this stage a large nucleus,

with a mass of central rounded or elongated chromatin, is observed, and the protoplasm is already vacuolated. After four days the parasites have become larger, the vacuoles more distinct, and the pigment less in quantity and scattered. After six days the parasites are very large, and are seen in the thickness of the middle intestine bulging into the body cavity, from which they are separated by the external coat of the middle intestine. Some of them seem to have slight enlargements, and a great number of small bodies can be distinguished in them, consisting of a minute portion of nuclear substance around which there is a small quantity of protoplasm. These sporoblasts become so many sporozoites. There are, in addition, to be observed shining corpuscular elements of a fatty appearance which could partly be seen in earlier stages. After seven days the parasite contains an enormous number of filaments disposed radially around several centres. These delicate filaments (sporozoites or mastigopods) are 14 micromillimetres long. In some individuals a clear homogeneous mass is to be distinctly observed; in some two or three such masses can be seen; in others none are visible. Pigment still exists, and is found in the clear masses. From the parasite the delicate filaments escape through the burst capsule into the body cavity.

On the following days there are the burst capsules still found adherent to the intestine, and near them the sporozoites. These spread through the body cavity, and at a later stage are found accumulated in great numbers in the tubes of the salivary glands, either in the cells or in the duct. In this stage the capsules or the remains of them may still be found in the intestine, but in some mosquitos the remains of them are no longer to be seen. In the capsules and in the salivary glands the sporozoites were immobile, but in one case they saw them moving.

The process is the same as that observed and described by Ross in the proteosoma of the grey mosquito, and is common in many sporozoa. It consists essentially of increase of volume, accompanied by capsulation, which ends on the sixth day by division of the cell contents into very numerous small nuclei around which a little protoplasm is arranged, leaving a *nucleus de reliquat*.

In tertian fevers it is the mature non-sporulating forms which develop in anopheles, the body of the parasite being paler, less refractive, and a little larger at the same stage of development, as compared with the crescent. The pigment is less in quantity and finer. The first phases of life in the intestine of the mosquito thus show a distinction between the crescent and tertian. The full-grown capsulated parasite is about 70 micromillimetres in size.

In rare cases in anopheles taken from malarial houses and from stables, they found in the developing parasite, bodies of a varied form and length—some sausage-shaped, longer than a sporozoite, and with swellings, others about half the measure of a sporozoite, ovoid, straight, or curved. These bodies have a strong membrane of a yellowish brown colour, and contain a body comparable to a sporozoite, particularly conspicuous in the shorter forms. The various stages of the development of the membrane can be followed.

They are found in the midst of granular masses, which may or may not be capsulated, and are evidently analogous to spores which are found in other sporozoa, but they have not yet been able to trace their development. These bodies are of a similar nature to the "black spores" described by Major Ross.

Bignami and Bastianelli describe minutely the mode in which the parasites of tertian fever in developing in anopheles can be divided into two kinds, a smaller, *mikrogamete*, containing the male element, and a larger, *makrogamete*, containing the female element. Although no one has yet seen the entrance of a flagellum into a *makrogamete*, nor has followed the first consecutive nuclear modifications, they believe that the sporozoon which develops in anopheles is the fertilised *makrogamete zygote*. This view is based on structural resemblances to allied sporozoa in which sexual phenomena have been observed.

The Italian observers describe some other appearances which, as observations of this nature are sure to be extensively carried out in many parts of the world, it may be useful to describe. In specimens of anopheles which have bitten malarial persons for some time, there are frequently found in the salivary glands cells which, instead of being purely hyaline, contain the following appearances. Sometimes the whole cell is filled with minute bodies apparently rounded or slightly elongated, massed in heaps. In other cells they occupy only the centre part. Sometimes in a tube only a few cells are found containing these bodies, but more frequently one or more of these tubes are full of them. If the gland is ruptured these fusiform bodies escape. They are much shorter than a sporozoite, are finer, and are furnished with what resembles a nucleus. In some cases they find in the same cell, in the midst of these bodies, filaments which can be easily recognised as ordinary sporozoites which come from the capsules contained in the body cavity. They were at first disposed to consider these bodies to be a retrograde process in sporozoites which had not been expelled from the salivary glands, but in a later paper they describe that they found similar appearances in the salivary glands of newly-hatched specimens of anopheles, and that they can be referred with certainty to a process of secretion of the glandular cells and partly to the result of the method of preparation of the specimens. In addition to the bodies that have been described they have found surrounding the intestine and the dorsal vessel tubular or jar-shaped masses of spores which apparently belong to sporozoa. Usually a certain number of them have a yellowish-brown-colour which resembles that of the yellowish-brown bodies which have an undoubted relation to malaria, and the various stages of the formation of the dark membrane can be followed. After many examinations, in which they never found that they were derived from the parasite of malaria, they now believe that these are parasites *per se*, and are not parasites of malaria.

They had previously described in the developing eggs of some mosquitos numerous bodies which they thought might be interpreted as the spores of the human hæmatozoa, but as after further study they had not been able to meet with any intermediate stage connecting the parasite of malaria with the spores with

eight sporozoites which they had discovered in the eggs, they consider that these also represent an independent parasite.

The discoveries of Kilburn and Smith, by which it was shown that the hæmatozoon which causes the Texas fever of cattle is transmitted from the tick, which is its temporary host, to a second generation of ticks, by which it is again conveyed to cattle—the resemblance between the hæmatozoon of Texas fever and the human malarial parasite being so great that the proof that the one was conveyed by an insect was considered almost a proof that human malaria must be similarly conveyed, even before it had been demonstrated that it is so—rendered it necessary to investigate whether in anopheles also the parasite is transmitted from parent to offspring. The Italian observers have, therefore, made a diligent examination of this question. They cultivated the larvæ of anopheles taken from houses that were infected with malaria, and when the new generation of winged insects was developed they allowed them to bite healthy people. So far these experiments have turned out negative. At the Hospital Santo Spirito a person who had never had malaria exposed himself voluntarily to the bites of newborn anopheles. He was bitten from March 30 to April 20 by numerous insects of this species, which were developed in a room in the hospital. He has nevertheless continued to enjoy good health. In the Laboratory of Comparative Anatomy, Professor Grassi and five persons belonging to the laboratory exposed themselves to the bites from numerous anopheles developed in the laboratory from larvæ or nymphs taken at many parts of the Campana, from places near dwellings in which malaria had run riot. The first bites dated from April 10. These five persons continued to allow themselves to be bitten daily without one of them becoming infected with malaria. So far (up to May 7) all these experiments had been negative, and up to that date only those anopheles that had bitten human beings suffering from malaria had been shown to be capable of propagating the disease.

There is a strong presumption that the yellowish-brown spores found in anopheles infected by human malaria are equivalent to the black spores described by Ross in the grey mosquitos which have bitten birds infected with proteosoma, and that they are in the first stage of a phase of existence of the hæmatozoon which has not yet been discovered. Their strong membrane suggests a capacity to resist external agents, and it is natural to suppose that they might be destined to infect the offspring of mosquitos or man direct through drinking water. The harmlessness of the bites of young anopheles appears to show that they do not affect the offspring, and other experiments indicate that they do not cause malaria through drinking water. One of the experimenters has swallowed a great many of these bodies at various times without any bad effects. These negative results have raised the question in their minds as to whether it may not be possible after all that these black bodies may be degenerating forms, an idea which seems to be strengthened by their irregular forms and size and their stratified appearance.

ANOPHELES CLAVIGER: ITS HABITS.

It will be seen from what I have related of the experiments of the Italian observers that the habits of *anopheles claviger* are matters of enormous importance to the human race, and it is interesting to know that these insects pass the winter in human habitations, and that during this period their habits of migration are so much in abeyance that, although the *anopheles* in the dwelling-houses are found charged with human blood and contain the hæmatozoa of malaria, those in the adjoining stables and henhouses do not contain the parasite. This has, of course, an important bearing on the question of prophylaxis—a wide question about which you will probably hear more in the course of the discussion, which is to be introduced by Dr. Nuttall in the absence of Major Ross.

ANOPHELES: SPECIES.

In order to understand the bearing of Grassi's investigations, it is necessary to bear in mind that the family of European Culicidæ can be divided according to Ficalbi into three genera based on the following characters. The first *anopheles* (*anopheles*, *hurtful*), has the palpi in both sexes about the same length as the proboscis; the second, *culex*, the palpi about the same length as the proboscis in the male only, in the female much shorter; the third, *aedes*, the palpi in both sexes much shorter than the proboscis. Five species of *anopheles* have been admitted for Europe. The first three, without spots on the wings, are *A. bifurcatus*, *A. villosus*, and *A. nigripes*. The remaining two have the wings spotted—*anopheles claviger* (*maculipennis*) and *A. pictus*. Ficalbi thinks it is possible that the first three belong to one species, and Grassi states that from an examination of a certain number of individuals collected from different sources he is of opinion that *anopheles nigripes* is simply a variety of *anopheles bifurcatus*. This reduces the species in Italy to three—*anopheles bifurcatus*, *anopheles claviger*, and *anopheles pictus* or *pseudo-pictus*. Grassi, Bignami, and Bastianelli have now extended their investigations to all the three species, and have found that the parasite of malaria develops in all of them, although for Italy *A. claviger* is by far the most important.

ANOPHELES: GEOGRAPHICAL DISTRIBUTION.

The geographical distribution of *anopheles* thus becomes a matter of importance. The *anopheles claviger* is widely diffused in Europe. It occurs in Scandinavia, Austria, Germany, Russia, Italy, and the adjacent islands, and both *anopheles bifurcatus* and *anopheles claviger* were described in 1825 by Stephens as existing in England. It would be interesting to know what are its limits of distribution in England at the present time, and whether it can be found in districts in which malarial fevers still occasionally occur.

In the list of mosquitos contained in *Bulletin* No. 4, New Series, U.S. Department of Agriculture, Division of Entomology, no fewer than seven species of *anopheles* are mentioned as existing in the United States. Amongst them are *A. punctipennis*, *A. claviger* and *A. nigripes*.

SCHOOLS OF TROPICAL MEDICINE.

The same year that has seen the momentous discovery of the intermediate host of the parasite of human malaria has seen the establishment of schools for tropical medicine in Great Britain, of which the interest and importance will, I hope, not be confined to these islands. The material requisite for the study of tropical diseases has long been present in this country, and there has never been a time when it was not important to use this material for clinical instruction. It has been long utilised for the training of army surgeons, but no sufficient systematised instruction was provided for civilian medical men destined to practise in the tropics. Dr. Manson called attention to this deficiency with convincing force, and Mr. Chamberlain, bringing to the subject the clear perception and decision for which he is distinguished, took a step which at once brought the subject prominently before not only the profession but the people of England.

Now that so much has been done to further the study of tropical medicine in this country within one short year, we wonder why it was never done before. That this movement will not only be sustained, but that its scope will be largely increased, seems to me a matter of certainty. The imperial idea, which has now got a firm hold of so many people in this country, means such an expenditure of life and health in tropical countries, that it is a question whether the population of these islands is able to bear it. It is all the more necessary, therefore, that we should be able to provide for these countries a supply of thoroughly trained medical men. Two schools have in the meantime been established, one in London at the Seamen's Hospital, and another at Liverpool, and we may confidently expect excellent work from both. The circumstances connected with the establishment of the school at the Albert Docks are too fresh in your memories to require comment from me. The cynic might remark that the consecration of the school has been associated with an imitation of the barbarous rites performed in some semi-civilised countries where the gods are propitiated at the building of a temple by burying victims under the foundations. The establishment of the new school may be figuratively said to have had its victims. It is not necessary for me to dwell further on these unfortunate circumstances, nor to explain the metaphor. We have only now to express our good will to the new school, and we have every reason to believe that the investigations and researches which will be carried on in its wards and laboratories will bear excellent fruit. A centre of investigation into the ætiology and pathology of tropical diseases within easy reach of London is a subject of congratulation, for there are students and investigators to whom residence in London is a necessity, and who would not be able to utilise a medical school situated in the provinces. But, after all, London does not offer the greatest opportunities for the study of tropical diseases, particularly as regards clinical instruction, that exists in this country. Liverpool, with its immense shipping and its intimate relations with the African coast, supplies more patients suffering from tropical diseases than are to be found in London. It is very fortunate

then that private munificence and the enterprise of the University at that port have established a system of instruction in tropical diseases which an ample supply of patients and skilled teaching promise to render of great utility, the close association at Liverpool of instruction in tropical disease with the medical and pathological teaching of the University being an advantage which is too evident to require comment.

TROPICAL DISEASES AT NETLEY.

But if we take into account the number of patients suffering from tropical diseases, and the consequent opportunities for clinical study, the great military hospital at Netley stands out prominently as offering conspicuous advantages with which London and Liverpool cannot compare. The large and swift transports that are employed by Her Majesty's Government bring regularly sick soldiers—from India particularly—to Netley Hospital, and the number brought is so great as to render the opportunities for treating tropical diseases at Netley unique. Not only can no institution in this country be compared with Netley as a school for tropical medicine, but no other possible school in Europe could be compared to it.

The Netley Medical School is, I believe, due to the initiative of Mr. Sidney Herbert (Lord Herbert of Lea), who had been chairman of a Royal Commission to inquire into the sanitary condition of the army. The school was organised and opened in 1860. Four professors were appointed to instruct in military hygiene, military surgery, and military medicine and pathology, and arrangements were made for practical and clinical teaching in these branches. The men originally appointed were Dr. E. A. Parkes, Sir Thomas Longmore, Surgeon-General Campbell Maclean, and Sir William Aitken. The Chair of Military Medicine was understood to be mostly concerned with tropical diseases, the invalids admitted from India and other tropical stations serving as subjects of clinical education. The school was transferred to Netley Hospital in 1863. The Chair of Military Medicine was held by Maclean till 1885; since then it has been filled by Dr. David Boyes-Smith, H. Cayley, and is occupied at present by our Vice-President, Lieutenant-Colonel Kenneth MacLeod. These names are an ample guarantee for the thoroughness with which tropical medicine has been and will be taught at Netley.

It is sufficient to mention that the teaching of pathology is in the hands of Professor Wright to know that that branch of medical instruction is in the hands of a master of modern scientific methods. As a matter of fact, those who have had the pleasure of meeting the young military surgeons who have passed through Netley cannot have failed to be struck by the thoroughness with which they have been instructed in the pathology and clinical management of tropical diseases.

That a school with the advantages of Netley should have been confined to army surgeons in a country whose connection with the tropics is so close as ours would be a subject of astonishment if we did not consider the manner in which education has hitherto been managed in this country. Private enterprise

has always to a large extent taken the place of Government regulation. That private enterprise by quickening competition, and by a process of survival of the fittest, has its merits and has, as regards general medical education, done well, cannot be doubted, but there are occasions in which private enterprise does not find its scope, because it is limited and cramped by service regulations and *esprit de corps*. It is hardly possible that if the advantages of Netley as a School for Tropical Medicine had been brought before the notice of the Government they would have refused to open its wards and its class rooms to civilians who intended to practise medicine in the tropics. Not only would no Government have taken the responsibility of refusing such a request, but, if the facts of the case had been sufficiently made known, I believe there is enough statesman-like initiative in Government circles to have led to spontaneous action on their part. Probably in no other European country would such opportunities be overlooked. The time which most young medical men can give to acquiring special professional knowledge after they have qualified in medicine is necessarily limited. It is of great importance, therefore, that as many cases as possible should be utilised in their teaching. Although for the purposes of scientific investigation a few cases may be sufficient, and the scientific work of a school may be not only not in proportion to the number of patients, but may conceivably be in an inverse ratio with the clinical instruction which is the main object of a school for tropical medicine it is quite different. From the time the young practitioner arrives in the tropics he will in most cases be left to work unguided. Clinical knowledge does not come by intuition, and cannot be got from lectures and theoretical teaching. It is of the utmost consequence, therefore, that the young practitioner preparing for the tropics should see as many cases as possible within the short time that he can devote to their study.

BRITISH MEDICAL ASSOCIATION.

SECTION OF TROPICAL DISEASES.

Wednesday, August 2, 1899.

Dr. GEORGE THIN, President of the Section, in the Chair.

Abstract of Proceedings.

THE PRESIDENT'S ADDRESS.

THE ÆTIOLOGY OF MALARIAL FEVER.

At page 1 of this issue Dr. Thin's address will be found. Its perusal will well repay the reader, for Dr. Thin has stated fairly and distinctly the work done, by Italians and by Englishmen, in regard to the mosquito malarial theory. Grassi, Bignami and Bastianelli in Italy have confirmed in all points, and advanced in many, the admirable work carried on in Calcutta by Ross, working on the lines suggested by Manson. Ross's discoveries were communicated to the meeting in Edinburgh in 1898, by Dr. Manson, and now Dr. Thin gives in detail the scientific methods pursued in Italy. The last stage in the mosquito malarial theory may be said to have been reached and there remains only the details of evidence—of very great importance, however—to be dealt with.

THE RÔLE OF INSECTS, ARACHNIDS, AND MYRIAPODS IN THE SPREAD OF DISEASES DUE TO PARASITES. By Dr. GEORGE H. F. NUTTALL (Cambridge).

(a) *The Rôle of Insects, etc., in the Spread of Bacterial Diseases.* 1. Passive rôle. The domestic fly and allied species are chiefly to blame in this respect. Incapable of "biting," they may from the nature of the food they seek carry pathogenetic bacteria in their bodies or within their alimentary canal and deposit them on lesions of skin or mucous membrane, or on food.

It is possible, nay, probable, and in many cases scientifically proved, that anthrax, plague, cholera, typhoid fever, frambœsia, and Egyptian and "Florida" ophthalmia are so diffused.

(2) Active rôle. Blood-sucking flies may play a part in propagating bacterial disease. Clinical writers report that cases of anthrax, septicæmia, pyæmia and erysipelas arise in certain instances from bites of flies; but experimental evidence is all against this statement. Experiments made by the writer on animals with plague, anthrax, mouse-septicæmia and chicken-cholera all gave negative results.

(b) *The Rôle of Insects, etc., in the Spread of Diseases due to animal parasites.* Insects, etc., whilst serving as intermediary hosts may play: 1st, a passive rôle, when they are devoured by a host of the parasites they contain; 2nd, an active rôle when, as in the case of the tick in Texas fever, and various mosquitoes in malarious affections of man and animals, they inoculate into a host by means of their proboscis; 3rd, in filarial diseases an intermediary position is played by the mosquito as it infects itself by sucking the blood of the definitive host.

Insects, etc., without serving as intermediary hosts may play: 1st, a passive rôle, when they transport eggs of animal parasites and deposit them in food, e.g., eggs of *Tœnia solium*, *Trichocephalus*, *Ascaris lumbricoides*; 2nd, an active rôle, by carrying the diseased animal from one animal to another and inoculating the parasite, e.g., Tsetse fly.

A CASE OF BILHARZIA HÆMATOBIA.

By C. P. CHILDE, F.R.C.S.

Mr. CHILDE stated that he had a case of a female under his observation who was suffering from all the symptoms of bilharzia infection. It is rare to meet with the disease amongst females, no doubt owing to the fact that they seldom are exposed to it as men are, but this patient, a white girl, aged 16, had up to two years ago, resided in Natal, where she frequently bathed in a pool of fresh water. Mr. Childe is of opinion that the parasite gained entrance to her body either by the skin or urethra, and asked whether any known drug or treatment could be suggested as a remedy.

Dr. MANSON discredited the suggestion that the bilharzia gained entrance by the skin or by the urethra, and Colonel MACLEOD said that, although drugs were useless to cure the disease, many persons suffering from bilharzia had reached old age.

FILARIAL PERIODICITY, by Dr. PATRICK MANSON, LL.D.

The singular phenomenon of the embryo filarial parasites appearing in a countless swarm in the cutaneous circulation during the night and disappearing from it during the day, suggests the questions:—

(1) What is the object of it? (2) Is it constant? (3) What becomes of the young filariæ during the day?

The answers to the first question is in all probability that the nocturnal appearance of the filariæ at the surface of the body is an adaptation of the habits of the parasite to those of the mosquito, its intermediate host. The answer to the second question is distinctly in the affirmative—filarial periodicity is constant. The third question "What becomes of the filariæ during the day?" Dr. MANSON is enabled to answer by a recent experience. A man suffering from filaria nocturna in his blood committed suicide by poisoning at 8.30 a.m. The hour is important, as about the time he died (he died almost instantaneously) the filariæ had retired from

the peripheral circulation for the day. At the *post-mortem* examination, 17 adult filariæ *Bancrofti* were found in a varicose lymphatic mass occupying a large part of the pelvis and abdomen. A hurried microscopic examination made at the time gave the following results. In the blood of the lung, a pulmonary artery and a pulmonary vein, embryo filariæ were met with in enormous numbers. A large number were found in clots of blood in the left ventricle of the heart and in the aorta. A few filariæ were found in the coronary artery and vein, in the right ventricle of the heart and in a middle cerebral vein. No filariæ were seen in the bone marrow, in the spleen and in the liver. Subsequent examinations of the blood films after fixing and staining gave almost similar results. The number of filariæ in the blood expressed from the lung was prodigious, as many as 80 or 40 being visible in some fields of $\frac{1}{4}$ inch objective. Dr. Manson concludes from these observations that filaria nocturna, during its temporary absence from the cutaneous circulation, is present in the large blood vessels, particularly the arteries, and a few in the capillaries of the muscles, brain, kidneys, and heart muscle, but that the vast majority are lodged in the blood vessels of the lungs. By what mechanism the filariæ maintain their position in the blood stream of vessels such as the aorta and carotids it is impossible to say.

Thursday, August 3, 1899.

COLONEL KENNETH MACLEOD, M.D., Professor of Military Medicine, Netley, Vice-President, in the Chair.

PSILLOSIS OR SPRUE; ITS RELATION (ÆTIOLOGICAL AND PATHOLOGICAL) TO OTHER FORMS OF TROPICAL DIARRHŒA, AND ITS TREATMENT.

The discussion was introduced by Dr. THIN, the President, in a paper entitled, "*Notes on Psillosis or Sprue.*" Dr. Thin divides cases of sprue into four groups:—(1) In Sprue patients from the Far East (Eastern Archipelago), the mouth, tongue and throat symptoms are early prominent, with supervening diarrhœa; (2) Patients who contract Sprue in India exhibit copious, watery stools from the first; the mouth and tongue symptoms not developing until a later stage of the disease. In Sprue patients from the Far East mouth and throat symptoms are very great, long before the disease has led to much debility or emaciation. The reverse is the case in Sprue patients from India suffering from diarrhœa alba, or white flux; for with them, "mouth" symptoms are usually synchronous with advanced malnutrition and emaciation. (3) A form of chronic diarrhœa occurs in elderly persons or in persons of long residence in the East. It is characterised by looseness of the bowels, gradual thinning of the epithelial covering, and considerable rawness, of the tongue. (4) In persons who have been in the East intestinal symptoms may develop long after they have returned to Britain. In reference to the pathology of the disease little is known. We find there is a general atrophy of tissues and organs, that there is a special connective tissue development—a true sclerosis—of the submucosa of the intestine, more especially of the ileum. The cause of this irritation is unknown, but that the irritation may be sufficient to produce an enteritis was shown at the *post-mortem* of a patient, who died from a very acute attack before emaciation had occurred.

The ulcerations described by Bertrand and Fontan as occurring in the intestine Dr. Thin is inclined to consider of secondary consequence. As regards ætiology, it would appear to be a specific disease associated with residence in certain parts of the world and absent in others. The poison, whatever it is, alters the secretion of the small intestine and allows the food bolus to retain its acidity; it destroys the colouring matter of the bile, and seems to develop freely when farinaceous and animal foods are given. The poison would appear to be starved out by limiting the patient's diet, or when the food consists exclusively of milk, and, in some cases, by an exclusive diet of meat juice.

Dr. HENDERSON (Shanghai): In 1868 when Dr. Henderson began practice in Shanghai, no distinction was made between cases of chronic diarrhoea and the cases now known as "sprue." In Shanghai two types are met: first, patients of long residence in the East, who, during it may be twenty or more years, have enjoyed good health. In them a morning diarrhoea gradually develops, and a sore mouth, intermittent in character, supervenes. Secondly, patients after five to six years' residence may become the subjects of sprue. In these patients there is a previous history of bowel complaint occurring for the most part during the hot weather. During one of the occasional attacks of intestinal flux the mouth becomes sore and the diarrhoea becomes established. The diarrhoea and sore mouth advance together in sprue. Dr. Henderson stated that he had seen the mouth lesions remain after the stools had become solid as the result of treatment, but he had never seen sore mouth precede diarrhoea. Death in sprue is brought about by a process of gradual starvation. *Post-mortem* evidence shows that there is diminution in size and weight of nearly all the organs and tissues of the body. The liver is especially small, but perfectly healthy when cut into; the atrophy of the mucous membrane is apparent throughout the whole alimentary canal. It is impossible, of course, to say whether this is due to starvation or to the effects of the original cause of the disease. Independently of treatment by diet, milk diet more especially, Dr. Henderson had found a preparation, made by boiling together simaruba bark 8 ozs., cinnamon bark 1 oz., in three quarts of water until reduced in bulk to one quart, when 1½ ozs. of brandy are added, very efficacious. The dose of the mixture thus prepared is a wineglassful three or four times daily.

Dr. WATSON (Portsmouth) described the case of an English lady who contracted an intestinal flux in India. Shortly after her arrival in India she had a sharp attack of dysentery. During the commencement of the next summer the patient developed distinct symptoms of sprue, that is, within fifteen months of her arrival in Northern India. The characteristic stools developed whilst living at a low hill station in India. On a milk diet the symptoms disappeared, only to recur, however, when a more varied diet was resorted to. On leaving India on account of her condition she enjoyed perfect health on the voyage home, but two months after reaching England the disease recurred. By milk diet and rest in bed all symptoms again disappeared, but during a stay in South Europe (San Remo), whither she had gone to pick up, a relapse occurred. Subsequently the tongue became inflamed, raw and tender, and it would seem that true sprue had set in.

Mr. JAMES CANTLIE (London) stated that in the treatment of sprue and, in fact, in almost all cases of intestinal flux, he had ceased to administer milk. He trusted to a meat diet, commencing, if need be, with raw meat juice or scraped beef, exhibited frequently, even every half hour if the patient was very weak. Later, when the severe symptoms disappeared, finely minced beef, lightly and rapidly cooked, in quantities of 5 oz. at a time, are to be given thrice daily. A plain calf's foot jelly, to allay the feeling of hunger, is allowed the patient between meals and during the night. Rest in bed, a wet pack applied to the abdomen twice daily for two hours at a time, and fixed by a large binder (bath towel) tightly bound round the abdomen, are useful and essential parts of the meat diet treatment. By a milk diet a stool of deceptive hardness was induced. It was pale, non-fæcal in character, and was composed of cheesy masses of condensed curd of milk. A meat diet brought down a markedly bilious stool, helped to exercise the hepatic functions, and thereby increased the size of the atrophied liver. Milk relieved the symptoms, but by meat alone can a cure be effected.

Capt. LAMB, I.M.S., said the tendency of milk to form curd in the intestines might be thwarted by adding citrate of soda (1 in 400) to the milk.

Major GILES, I.M.S., doubted if he had ever seen a case

of sprue in Northern India which answered in all points to the disease described by medical men practising in China. He had seen many cases resembling the one described by Dr. Watson, but the general opinion in India was that they were merely hill diarrhoea. Major Giles believes that "hill diarrhoea" is a condition in which the hepatic function is suspended, and that is brought about by the treacherous chills of the hill climates in India, although it is in no way confined to hill stations.

Dr. MANSON (London) considers that sprue occurs anywhere in the tropical and subtropical zones, and he had met with cases from China, Japan, the West Indies, India, &c. It would appear that in the case of sprue and associated ailments we are dealing not with a single disease, but a group of diseases, caused, it may be by a variety of causes, as in the case of dysentery. Sprue was perhaps an example of disease attacking an organ or organs which had been exhausted functionally by the demands of climate. Dr. Manson stated that he had seen a case of sprue in which mouth and throat derangements were alone met with, diarrhoea not developing until some time subsequently. He did not consider that any one line of diet or any drug could be considered a specific in sprue; but that a diet consisting solely of milk, of milk with fruits such as bananas, of meat alone, or of a mixed character, should be employed as the condition of the patient would seem to justify. Rest, and above all warmth, are essential factors in the treatment of sprue.

Dr. MULICK (Bombay) stated that he had never seen sprue amongst natives of India. From experience gained in England in two cases of sprue he had observed great benefit accrue by using koumiss as a diet instead of milk.

Inspector-General TURNBULL, R.N., in reply to a question by the Chairman, Colonel MacLeod, said he had never seen sprue occur on board any of Her Majesty's ships; he had met, however, with a few cases of sprue in naval hospitals at home in men who had served on the China station. Dr. Turnbull described a case in which the meat diet recommended by Mr. Cantlie was attended by excellent results.

Dr. SAMBON (London) did not consider the differences mentioned in the seat of the lesions peculiar to sprue as of sufficient importance to justify dividing the disease into two classes. The geographical distribution of sprue seemed to point to sprue being a distinct disease. The specific germs, like those of blackwater fever and other diseases, probably become parasitic at an early period, but it is only under conditions of low health or metabiosis that the disease manifests itself. Thus may be accounted those cases which occur for the first time in Europe, but always after residence in the endemic regions of the disease.

Dr. RHO (Italy), stated that French observers in Cochin-China affirm that minute ulcerations and erosions occur in the lowest part of the ileum and the whole length of the colon. The same authorities also declare that traces of blood are always found in the stools of sprue patients at the beginning of the disease, and they regard sprue as a residue of a chronic dysenteric state. Dr. Rho said that the condition of the intestine met with in sprue resembled the condition of the stomach found in Reichmann's disease, which is characterised by dilatation of the stomach, sclerosis of the wall and great secretion of very fluid mucus (gastro-succorrhœa).

Colonel MACLEOD remarked that, in Northern India, the disease recorded by medical practitioners in China could scarcely be said to occur. Hill diarrhoea resembled in many points the signs and symptoms met with in sprue, but the "mouth" symptoms were never of the characteristic nature described by observers in China.

Capt. W. J. BUCHANAN, I.M.S., contributed a paper to the discussion on sprue in which he stated:—(1) that primary or protopathic sprue is common among natives of India; (2) that secondary sprue following in (a) dysentery and (b) acute enterocolitis or enteritis is common; (3) that incomplete or arrested sprue is probably very common; (4)

that the condition known as "famine diarrhœa" is essentially the same in its symptoms and ultimate results as sprue; (5) that in many cases of chronic relapsing dysentery a condition strongly resembling sprue is met with, and that characteristic frothy pultaceous diarrhœa alternates with the dysentery.

Dr. THIN, in reply, remarked that there was no evidence that the liver was diseased in sprue, or that the shrinking depended on anything more than the persistent diarrhœa and general wasting. The white stools passed with a milk diet do not consist of undigested milk. Milk in the earlier stages is the best diet; but in the latter stages of the disease a meat diet was efficacious.

A discussion arose concerning the treatment of sprue by santonin as recommended by Dr. Begg, of Hankow, but several speakers declared they had employed it without benefit.

SUPRA-HEPATIC ABSCESS.

Mr. JAMES CANTLIE (London) read a paper on the symptoms, pathology, and treatment of a disease which he defined as follows: By a supra-hepatic abscess is meant the formation of pus between the layers of the broad ligament of the liver, having as boundaries the peritoneum circumferentially, the liver below and the diaphragm above. The affection may be unattended by hepatitis, dysentery, or any other abdominal ailment. It is characterised by a sudden onset, symptoms of fever, cough, and some respiratory distress and lung congestion, and, when allowed to pursue a spontaneous course, usually terminates by the pus finding its way through the diaphragm and lung to a bronchus from which the purulent matter is expectorated. Mr. Cantlie objected to the term "liver abscess" being employed as the name for a definite lesion. He believes hepatic abscesses can be scientifically grouped under three headings—supra-intra- and infra-hepatic, differing in ætiology and pathology. Supra-hepatic abscess commences as a lymphangitis of the vessels between the layers of the broad ligament of the liver the result of chill; the inflammatory effusion, speedily ending in pus, caused an area of dullness on percussion between the liver and right lung in the neighbourhood of the right nipple line. The dull area is shaped like an inverted saucer or cup on the upper margin of the liver, with its highest point usually internal to the right nipple line. The pus, when withdrawn by an aspirating needle before the abscess burst, was invariably sterile. Mr. Cantlie considers an abscess in this situation to be of non-dysenteric origin, and affirms that the discussions which have so long been associated with the question of the dysenteric or non-dysenteric ætiology of hepatic abscesses are thus explained. He considers it improbable that abscesses arising from ulcerative lesions in the intestine could present a sterile pus, and is of opinion that the supra-hepatic abscess is the true "non-dysenteric" and "sterile pus" abscess. As regards treatment, Mr. Cantlie recommends early aspiration as a prophylactic, as a means of diagnosis, and as an indication of treatment. When pus is found, tapping through the thorax by a trocar and canula, and drainage by a large sized india-rubber tube, as recommended by Manson, are attended by better results than when incisions are made to reach the seat of the abscess.

Dr. MANSON remarked that the sterility or apparent sterility of liver abscess pus may be explained by the micro-organisms having died out. The amœba coli is often missed when examining pus from the liver, by the fact that the amœba does not occupy the centre, but lies on the wall of the abscess, and should be searched for in the discharge from the drainage tube some days after the operation.

Dr. SAMBON refused to recognise that pus from the liver could be primarily sterile. A collection of pus must have been caused by some kind or other of irritant. When an amœba is not found it is probably because it has been destroyed—in fact, the destruction of the irritant is the very object of phagocytosis.

Capt. LAMB, I.M.S., stated that not only were abscesses

containing sterile pus found in the situation described by Mr. Cantlie, but also in the liver substance itself.

Dr. RHO (Italy) stated that supra-hepatic abscesses were not a disease of tropical countries specially. Both supra-hepatic and supra-splenic abscesses are met with in temperate climates, and they depend upon some chronic disease of the intestine of an ulcerative nature.

Dr. E. CHARLES (Falmouth) attaches great importance to the presence of a leucocytosis as a means of diagnosis in suspected cases of liver abscess. He inclines also to the opening of hepatic abscess by way of the abdominal wall rather than by the thorax.

Dr. HENDERSON (Shanghai), spoke strongly in favour of the metal drainage tube recommended by Dr. MacLeod, of Shanghai, as being in less danger of getting nipped between the ribs, and thus occluded, than is the case with india-rubber tubing.

DYSENTERY AS A TERMINAL SYMPTOM OF DISEASE IN THE TROPICS.

Captain W. J. BUCHANAN, I.M.S., contributed a paper calling attention to a form of dysentery which supervenes in the later stages of many diseases in India. Captain Buchanan says, "as will be seen from the following 28 cases, this 'terminal' dysentery (as I call it) frequently supervenes in the last stages of many chronic diseases; it is a certain symptom of impending death, and may commence even three or four weeks before that event takes place." Of the 28 cases given in detail all had been in hospital for some other severe illness. The dysentery in most cases is of a low gangrenous type, but in some it is of a more acute nature. Captain Buchanan believes the condition is essentially a degenerative process and more allied to *noma* or gangrenous ulceration of the buccal membrane than to any one of the many pronounced types of dysentery.

Dr. MANSON, in his remarks on the paper, said that these cases illustrate the fact that in healthy conditions, when resistance is normal, one of the germs of the many forms of dysentery, as probably of cholera and other disorders, may be present in the alimentary canal and not cause any pathological effect, but when the individual is weakened from any cause the parasites may become pathogenetic.

Dr. SAMBON said that Captain Buchanan's cases of terminal dysentery were cases of mixed infection; the parasites in dysentery, as in black-water fever and sprue, &c., may be latent for long periods in the system, but given favourable conditions—low health, metabiosis—they may suddenly manifest themselves in typical attacks of their respective diseases. Colonel MacLeod had mentioned the special prevalence of terminal dysentery in leper asylums, and that the disease is also common in lunatic and other asylums in Europe. When collective dwellings become infected the disease may persist for years, as is the case with beri-beri and yellow-fever. He hoped the day was not far when terminal dysentery and complicating beri-beri would be as obsolete as hospital gangrene is now.

HÆMOGLOBINURIC FEVER AND PALUDISM.

Surgeon W. H. STALKARTT, M.D., R.N., read a paper on this subject, in which he stated that men on board ship on the West and East Coasts of Africa were seldom, if ever, attacked by blackwater fever. Again, that on the Zambesi and Shiré rivers a practical immunity of the troops obtained. The disease occurs only after prolonged exposure in regions where the disease is endemic. Besides intertropical Africa, blackwater fever is met with in Sicily, Sardinia, tropical America, New Guinea, and in the Indian Terai. In Australia and in many districts of India the disease is unknown. Surgeon Stalkartt related a case of blackwater fever in which quinine was given as a precautionary measure against malaria and in which blackwater fever did not recur. He stated that the circumstances which bear strongly against the malarial hypothesis of blackwater fever being malarial are the limited distribution of the malady and its

non-occurrence in certain highly malarious regions. Surgeon Stalkartt regards blackwater fever as a distinct disease, possibly malarial in nature, and does not believe that it is due to quinine poisoning.

Dr. MANSON grouped the arguments for and against blackwater fever being malarial as follows:—(1) Arguments for regarding blackwater fever as being a malarial disease: (a) It occurs in highly malarial districts; (b) the malarial parasite is often found in the blood; (c) the disease occurs in persons who have had many attacks of malaria; (d) prolonged residence and consequent malarial saturation are usually antecedent to an attack of blackwater fever. (2) Arguments against regarding blackwater fever as malarial: (a) Its limited geographical range as compared with malaria; (b) the malarial parasite is not always found, and even when found they are not always of the same kind; (c) it is not certain that the fevers preceding attacks of blackwater fever are really all of the true malarial character; (d) cases have occurred within a short period of arrival in a region where the fever is endemic; (e) the epidemic seasons of malaria and blackwater fever do not always correspond; (f) the disease is not amenable to quinine.

Colonel MacLEOD stated that, although acquainted with cases resembling blackwater fever in India, the symptoms developed as a sequence to malarial fevers, but that they were not to be confused with the true blackwater fever met with in Africa.

Dr. SAMBON said he believed blackwater fever to be a specific disease. Its greatest analogies were not with tertian fever or summer-autumn fever, but with Texas fever. Several authors had observed a small unpigmented parasite in cases of blackwater fever which corresponded to that of the redwater fever of cattle. The morphological differences between the parasites of Texas fever and those of the summer-autumn group were but slight, and the almost constant concurrence of some form or other of malarial infection with that of blackwater fever rendered the differentiation still more difficult. No multiple, rosette-like division had been observed in Texas fever, but only simple binary division. In blackwater fever the association of two parasites within the same corpuscle and their apparent connection had been noticed by Woldert and others. This would be an important diagnostic character if it were confirmed.

Major GILES, I.M.S., said that the cases of malaria complicated with blackwater fever met with in India were not attended by a pronounced mortality as in Africa, and it would appear that the resemblance does not extend beyond the alarming symptoms common to both, and that the diseases are really quite distinct.

Dr. RHO considers Sambon's theory of blackwater fever as the most satisfactory, and it has in its favour all the arguments which by analogy we can get from hæmoglobinuria, of cattle or Texas fever. Several Italian observers, Tomaselli, Murri, &c., have ascertained the fact that in some malarial subjects a very few centigrammes of quinine can give rise to an access of fever with hæmoglobinuria.

Mr. REES (London) stated that the seasonal differences of blackwater and malarial fevers was most marked at one station on the Niger, where, as the malarial curve decreased, the blackwater fever curve gradually increased. Mr. Rees said that his experience is against the idea that hæmoglobinuria fever is due to quinine poisoning. Quinine was taken largely in Nigeria as a prophylactic, and those who adhered to quinine most carefully seemed to escape blackwater fever. Mr. Rees stated that he had known cases of blackwater fever contracted within two or three months of arrival in Africa, and he is not of opinion that blackwater fever is either malarial or due to quinine.

Dr. THIN remarked that in addition to the case he had recently published in which a man dead of blackwater fever was found to have the parasite of malaria in the blood-vessels of his brain, and pigment in the spleen and liver, he had examined portions of liver and spleen in another case

and found pigmentary deposit in these organs sufficient to indicate an acute malarial attack.

Dr. MOULD mentioned that a patient under his care in hospital for a sprained ankle developed blackwater fever. He had no quinine for three weeks and no malaria.

Dr. PORTER has seen cases of blackwater fever in Liverpool from the West Coast of Africa. He would ask whether the fevers met with in Colon, Jamaica, Honduras and Brazil are not of the same nature. He regards blackwater fever not as a distinct disease, but merely as the name of a symptom.

ON THERMIC FEVER (SO CALLED SIRIASIS), WITH SPECIAL REFERENCE TO ITS ALLEGED MICROBIC CAUSATION.

Colonel MacLEOD, M.D. (Netley) discussed Dr. Sambon's view of this question, namely, that so-called thermic fever is not due to heat *per se*, but to a microbe called or recalled into existence and activity by excessive heat under certain circumstances—climatic, local and personal. The name *siriasis*, revived by Dr. Sambon and adopted by some, did not advance our knowledge or nomenclature. The word, derived from Sirius, the dog star, would seem to imply that sunstroke is more common during the dog days (July 3 to August 11), but such is not the case, at any rate in India. No positive evidence has been advanced regarding the existence of a special microbe in cases of thermic fever. It has been shown indeed by Stiles that the injection of blood, taken from subjects of the disease, into a healthy person does not induce any pathological disturbance; therefore excluding the presence of either infective microbes or toxins in the circulating blood. Dr. Sambon reasoning that a microbe is present is by a process of analogy and exclusion both of them liable to fallacy, and the constructive evidence he thus arrives at is very feeble. There is one analogy, however, he has neglected to draw, namely, the analogy of the effect of reduced temperature or cold. The chilling of the body is as dangerous as the heating, if not more so. Almost all the arguments advanced by Dr. Sambon can be employed here, *i.e.*, the phenomena caused by over-chilling are peculiar and definite, as are over-heating; the incidence of cold stroke has some resemblance to the incidence of epidemic disease, being in some instances single or sporadic, in others multiple, simultaneous or quickly successive; hygienic and dietetic conditions affect heat conservation as well as heat diffusion; the duration of attacks, many or few, grouped or otherwise, is precisely limited by the duration of excessive cold in one case and excessive heat in the other. The parallel is almost perfect and yet no one thinks of invoking a microbe to explain the effects of cold.

Dr. Sambon's argument by exclusion, Colonel MacLeod said, is also very weak. It takes this form; excessive heat, solar and artificial, can be endured *per se* by the human organism with impunity, therefore something in addition is necessary to render it noxious. Now, Colonel MacLeod said, experiments prove that animals exposed to great heat die with symptoms resembling sunstroke in man. These Dr. Sambon sets aside as inapplicable in man, but without reason or fairness. Dr. Sambon further excludes cases of heat stroke on the ground that they are due to defect in the subject rather than to potency of heat influence. This is not the case, as perfectly healthy people suffer from sunstroke. Colonel MacLeod takes the view of sunstroke put forward by Hertz and Wood. He also pointed out that just as hepatic exhaustion succeeds hepatic stimulation, so continued exposure to heat impairs the power of resistance to heat, and one of the principal sequelæ of heat stroke is an incapacity to endure or resist heat.

Dr. SAMBON said that the idea that *siriasis* was explicable only by microbic action was gaining ground daily, and he had every confidence that his opinion would be confirmed. Cagicol and Lapierre had described a micro-organism. He attached no importance to this parasite, which might or might not be the cause of *siriasis*. The strongest argument

was that of the peculiar geographical distribution of the disease. Heat and moisture were certainly important factors in the prevalence of siriasis, but that only within its endemic areas. American authors, and especially Ira Van Gieson, had studied very carefully the pathology of siriasis, and they were obliged to admit that heat alone could not account for the blood changes and morbid lesions of the disease. The only interpretation, therefore, open to them was that of the action of some toxic substance. Ira Van Gieson believed siriasis to be a species of autointoxication, but taking into account the geographical distribution of the disease, its occasional epidemic outbursts, its relapses, &c., we were bound to believe that the poison which induces the acute parenchymatous degeneration of siriasis could only be of microbic origin.

Major GILES said that the fact that sunstroke had a geographical distribution supported Sambon's theory. He had seen soldiers in Natal working at a temperature which in other countries would in all probability have induced sunstroke. In Natal, however, sunstroke was unknown.

Dr. WATSON said his experience was that men exhausted from want of food, and who took excess of alcohol, were most liable to sunstroke.

Dr. MANSON said some organisms thrive in high and some at low temperatures; but it must not be assumed that because sunstroke occurs at high temperatures a high temperature is the cause. The organism, if such there be, affects only certain parts of the world. This consideration is alone a very important one, and favours Sambon's idea.

Dr. THIN considers that direct sunlight, as distinguished from simple heat, has much to do with the causation of sunstroke. He considers that in many instances sunstroke was at least partly due to the direct effect of light through the eye.

Dr. RHO would divide sunstroke into two classes: the first, real sunstroke, occurs sporadically with no very high temperature; the second, epidemic in nature and accompanied by hyperpyrexia. The former is probably due to fatigue and toxins of the body; the latter, as contended by Dr. Sambon, is probably infectious and due to microbic infection.

Lt.-Colonel BOILEAU and Dr. PARKER also joined in the discussion.

Lt. Col. MacCartie, I.M.S., contributed a paper appertaining to the discussion on sunstroke, in which he stated that soldiers were more liable to heat apoplexy than any other class, because of the nature, texture and quantity of their clothing and because, owing to their uniform their clothes are so buttoned up and strapped down that the air cannot circulate about their bodies. He had seen men in mufti in India march alongside soldiers in uniform, and, although carrying heavier loads than the soldiers, they came in fresh and fit at the end of the march, whereas their brothers in uniform were utterly exhausted, and some of them suffered from heat apoplexy.

ANTIVENINE. By Staff-Surgeon O. W. ANDREWS, R.N.

This paper dealt with well-nigh the whole subject of antivenine; and a detailed account of a series of experiments performed by Major Semple, R.A.M.C., Major Lyons, I.M.S., and Staff-Surgeon Andrews, R.N., were submitted.

The composition of venom, Dr. Andrews pointed out, has been investigated by Dr. C. J. Martin, who says that venom consists of coagulable proteid and non-coagulable proteid, and that the coagulable proteids may be regarded as albumoses rather than as globulins, the albumoses being those known as hetero- proto- and deuto-albumose. The venom of snakes is not a simple poison, but is composed of at least two distinct poisons, one of these, the one which has the power of producing hæmorrhagic extravasation and intravascular clotting, is destroyed by heating a solution of venom to 85°C. (1°), but there is another or others not destroyed by heating to the temperature of boiling water (100°), 102°C. Boiling for twenty minutes completely destroys all toxicity of venom.

M. Calmette maintains that the action of antivenine serum is in no way a chemical reaction, but a physiological process by which the white blood corpuscles are stimulated to carry on intracellular digestion, which he regards as a variety of phagocytosis. M. Calmette considers that the venom is destroyed only within the leucocytes, and if the amount of serum injected before, with, or after the venom, has been sufficient to impregnate the leucocytes so that they can carry on this process of digestion, the animals will resist lethal doses of venom in the same way that animals vaccinated against specific diseases resist.

The dose of serum required to protect a man against a lethal dose of the most venomous serpents is from 10 to 20 cc., but additional security would be afforded by increasing the dose to 80 cc. or even 40 cc. No ill effects attend the injection of the serum.

Mr. REES said that a statement made by Dr. Andrews he could confirm, namely, that one of the two toxins of venine produced intravascular clotting. He had seen two fatal cases in Nigeria in which marked thrombosis of the large veins of the bitten limb had occurred.

MALARIAL OR CLIMATIC NEURALGIA AND ITS TREATMENT.

By A. S. FAULKNER, I.M.S. (retired).

The symptoms of this affection referable, especially, to the nervous system are:—The regular periodicity of the attacks, acute if not excruciating pain in the set of nerves implicated (usually the facial), the pain being characterised by being confined only to the one set of nerves, but shifting from one nerve to another in that set. At first the pain is accompanied by a slight rise in temperature, followed by copious perspiration at the height of the attack, which is indicated when the pain has reached its acutest stage. Palpable throbbing of the arterial system in connection with the affected nerves is always evident, and a feeling of nausea or actual vomiting takes place. The attacks are followed by marked general depression and subsequent lassitude. Quinine properly administered is a specific in the treatment of such cases.

THE GEOGRAPHICAL DISTRIBUTION OF UNDULANT (MALTA) FEVER. By CAPTAIN M. LOUIS HUGHES, R.A.M.C.

The geographical limitation of the endemic prevalence of this fever appears to be primarily a question of temperature. In Europe, north of the annual isotherm of 55° F. (about latitude 46° N.) only imported cases are met with which do not start endemic foci affecting others.

South of this line the endemic prevalence of the disease becomes of increasing importance until we reach the annual isotherm of 60° F., where we find the disease firmly established, especially prevalent in those months during which the mean temperature is at or above 60° F.

A FURTHER CONTRIBUTION TO THE STUDY OF MALARIAL EYE AFFECTIONS. By Major M. T. YARR, R.A., M.D.

Conjunctivitis.—At least three different varieties of conjunctivitis have been found connected with malaria, viz.:—(a) Intermittent ophthalmia; (b) conjunctival injection due to neuralgia of the fifth cranial nerve; (c) epidemic conjunctivitis.

Keratitis.—The forms of keratitis ascribed to malaria are:—(a) Dendritic keratitis; (b) keratitis profundus; (c) vascular keratitis (herpes corneæ). Major Yarr also mentioned a case of iritis, a case of cataract and a case of monocular ciliary spasm associated with aphasia, all of which were distinctly malarial in origin.

The section was well attended and the interest taken in the debates and discussions most marked.

The proceedings terminated with a hearty vote of thanks to Dr. Thin, the President, and to Fleet-Surgeon J. Tyndall, R.N., and J. R. S. Robertson, M.B., the Hon. Secretaries.

OBSERVATIONS ON THE ÆTIOLOGY, DIFFERENTIAL DIAGNOSIS, AND TREATMENT OF BERI-BERI.

By P. T. CARPENTER, M.R.C.S.ENG.,
Colonial Medical Service.

THE following is an abstract from the same notebook from which I extracted my paper, "The Clinical Aspects of Beri-Beri," which appeared in the July issue of this JOURNAL.

Fearing, however innocently, to serve up a mere hash, as it were, of recent literature on the subject, which, I think, is not uncommonly done, I have adhered as strictly as possible to my observations as they appear in my original notes. This fact is my apology for thus venturing to submit this second article.

The origin, indeed the meaning, of the word *Beri-Beri* is obscure. It is said by some wiseacres to be derived from the Malay-Japanese, meaning stiff-gait. Others that it is the Malay word *Biribiri*, a sheep; again, that it is derived from Sanscrit, *Bhāra*, a load or weight. In Japan the same disease is called *Kakké*, which bears a strong resemblance to the Malay word *kaki*, a foot. One thing, however, is certain, and that is that the name has been applied to more than one disease. Many times patients have told me that they had Beri-Beri (a name well known to most natives in the East) when they were obviously suffering from acute elephantiæsis, thrombosis of veins, renal or cardiac dropsies, the œdema of impoverished blood, e.g., malarial cachexia, scurvy; in fact, anything in the way of swollen feet or legs is dubbed Beri-Beri in countries where this disease is common. There are, however, other diseases, as alcoholic paralysis, general spinal paralysis (sub-acute anterior poliomyelitis), and anchylostomiasis, which do bear a marked resemblance to Beri-Beri. Indeed, it was but recently considered in Ceylon that anchylostomiasis and Beri-Beri were one and the same. There is no doubt, however, that the disease about which I am writing is a distinct disease, though very closely allied to other forms of peripheral multiple neuritis. Different localities may possibly have their different varieties, for cases which I saw in Singapore were not quite the same as those I treated in North Borneo. It has, I believe, been stated that this disease occurs mostly on the eastern seaboard of countries in the Malay Archipelago. This must be a mistake, I fancy, probably due to the fact that the east coasts are more densely populated, and consequently more of the disease is apparent.

The cause of the disease is at present undetermined. It is certainly not the *dochmius duodenale*, for this parasite is, as often as not, conspicuous by its absence, as shown at *post-mortem* examinations of Beri-Beri cases, and by its frequent absence during the life of the patient, proved by the negative results of examination of the *fæces*. In many *post-mortems* of Beri-Beri cases of course it is found, for the anchylostoma is almost as common in the tropics as the *ascaris*. Out of half a hundred or more autopsies of Beri-Beri cases I have never, however, discovered the parasite of anchylostomiasis in sufficient numbers to account for even serious symptoms, much less death.

Various food-stuffs have from time to time been said to have caused the disease; pork has been particularly maligned on this account, probably because the muscular tenderness of trichinosis has been mistaken for tenderness of Beri-Beri. However, it is absurd to even discuss the matter, for are not the Malays and Japanese subject to Beri-Beri, and are they not prohibited by their religion from eating pork?

Rice has been accused also; and it is a fact, certainly, that the majority of the victims of Beri-Beri are those whose staple food is rice—people who generally eat nothing but rice, with the addition of a little dried fish. It is found wise to eschew rice in the dietary of Beri-Beri patients, but principally on account of the distension of the stomach which it is liable to cause, and for the fact that it is not a sufficiently nutritious or nitrogenous diet. Rice has never, as far as I am aware, been proved to be a cause of Beri-Beri, nor has it been disproved to be so. But the onus of proof surely rests with those who advocate this rather far-fetched theory. The unsatisfactory argument of the beneficial result of the deletion of rice from the rations in the Japanese navy is too threadbare to stand repetition. Other foods, too, have been thought to cause the disease, but until more satisfactory proofs are forthcoming it would be idle to discuss them.

Overcrowding has been looked upon as one of the chief causes of Beri-Beri. My own experience does in no way support this view. That overcrowding may assist the disease when once started seems possible; to say that it can cause it is another story altogether. One might as well say that overcrowding, *per se*, could cause cholera. For example: It frequently happened, unfortunately for the planters in British North Borneo, that healthy and strong coolies (mostly Chinamen newly arrived from seaports of China) ran away from the estates, and lived in the jungle for days at a stretch, sometimes, though not often, weeks. If they did not die in the bush, which they often did, they were invariably brought back, for a Chinaman is not often able to make his way through dense tropical jungle. In almost every such case that came under my notice the poor wretch returned sick, and developed Beri-Beri within a few weeks. Such surely are not overcrowded in the jungle! They may have eaten poisonous berries, or drunk unwholesome water, and probably did both. But if this unusual diet produces the disease we should have to expect only runaway Chinamen to get Beri-Beri. I need not add this would be absurd. These men were not overcrowded certainly, but they were exposed to worse influences: they were exposed, day and night, in the open air, in a state of semi-starvation, to the exhalations from a damp jungle soil, in a region where Beri-Beri was endemic, and most soils are known to harbour pathogenic organisms, and why not the germ of Beri-Beri?

Again, I had at one time under my medical supervision in Borneo a shed which I used exclusively as an ulcer hospital for the large sloughing ulcers which are now called, I believe, tropical sloughing phagedæna. The shed was built on piles some three feet off the ground, and accommodated twenty

patients, and could have easily taken thirty. It was well floored to prevent, as far as possible, miasm rising into the ward. The walls were really no walls at all; they were made up of a series of movable shutters or blinds, which were only closed when it was necessary to keep out wind or rain.

Practically, then, these patients were undergoing "open-air treatment," as is now advocated for phthisis. There was an abundance of fresh air, no possibility of the accumulation of foul gases, &c., certainly there was no approach to overcrowding, nor was there any previous infection of the building, for it was only used for six months, and was newly built for the purpose. It was short lived for this reason: half the number of the patients admitted (for phagedenic ulcers) died of Beri-Beri within six months! There were fifteen in all that died. None were sick (except for their bad ulcers on the legs) on admission. These were not overcrowded. They were well looked after in every way; but, a fatal exception, they were exposed too much to the influence of the "ground air," the miasm was not properly kept out of the ward, and gained access to the patients, carrying with it, we may presume, the organism of Beri-Beri. Probably anything which sufficiently lowered the vitality would predispose to Beri-Beri—prison life, for instance, and especially prison life in an Oriental gaol. I believe, however, I have good grounds for thinking that there is something, a germ of some sort probably, in the exhalations of the soil (miasm) of districts where Beri-Beri is endemic, that may actually, *per se*, cause the disease in subjects predisposed. The natives in some tropical countries appear to be instinctively aware of this, for they guard themselves from miasm as far as they can: they build their houses on long poles, and preferably over the water. That the Dyaks, Malays, and Sulus of North Borneo do thus build I can vouch from personal knowledge. Europeans have intuitively followed the example of the natives in this respect, and wisely. As one might expect, they have improved on and extended this principle by cementing their basements, &c. The average planter could not give a satisfactory explanation, probably, as to why he builds after the native custom. He has a vague, but correct idea that to sleep near the ground is dangerous. It is for this reason, together with the many little comforts he can command, that the European is not frequently attacked with Beri-Beri as the plantation coolie, a fact which I am aware some may doubt. At any rate, it is my experience that this disease rarely attacks people who can afford to surround themselves with necessary comforts, and avoid over-exposure to miasm, as Europeans invariably can in the East. For example, I may add that during the two to three years I had sole medical charge of some twenty-five European planters in British North Borneo only two cases of the disease occurred amongst them (the brief notes of which, as they appear in my books, I append), and both cases were mild in the extreme.

The native servants of Europeans also are comparatively safe, as far as my experience teaches. It is the poor man, the common labourer, who is the principal victim of Beri-Beri—the ill-housed, the poorly fed, and oftentimes, the overworked coolie.

For, although I am perfectly aware what the planter-managers would say to this—"Oh, dear no! It is the hard-working coolie who does not get sick"—the man who appears to do very little work is frequently overworked, and we, as medical men, cannot overlook the fact that the weakly one may be overworked with the lightest task, and suffer abuse besides for being thought to be a lazy vagabond. It is often the onerous duty of the medical officer of the estates to protect such, although he may be sorely tempted to treat them as malingerers. The manager's "hard-working-and-best coolie" is generally the exceptionally strong man, the survival of the fittest. He is not often overworked. Yet he, too, is attacked sometimes.

Finally, there is one very important and very peculiarly remarkable point to be considered in the ætiology of Beri-Beri. It is that prolonged periods of inactivity, and surgical operations involving serious wounding, are liable, with apparently no other cause, to terminate in Beri-Beri. This was so well recognised in Borneo some five years ago that I well remember, when I had an unfortunate gun-shot accident, which laid me up in bed for many weeks, the great fear of my friends, and I might add of myself, was that Beri-Beri would supervene. Fortunately for me it did not. But in similar cases I have known it to. The cases of the patients in the ulcer hospital above mentioned might be considered as examples. I recollect four consecutive major amputations (of lower limbs) terminating thus in Beri-Beri. Although the stumps healed perfectly, the patients never left the hospital alive; they died of Beri-Beri. Indeed, so certain, at one time, was this sad termination of operation cases that the fact had to be taken into very serious consideration before operating. I can offer no further explanation than the possibility of the poison being absorbed by the raw surfaces, although I must say I think it most unlikely, and especially when antiseptic dressings are used, as they were in my cases. Personally, I have never known Beri-Beri to show itself as a contagious disease; my hospital servants mixed freely with patients with immunity. Patients are not generally isolated, except maybe for convenience of treatment. In concluding this portion of the subject I might add that I have never seen a child suffer from Beri-Beri.

The differential diagnosis may be very difficult. As mentioned before, many diseases have been confounded with Beri-Beri. It will only be necessary to consider the few which are more likely to be so mistaken. First in importance in this respect is alcoholic paralysis. Like Beri-Beri, this is a condition of peripheral multiple neuritis. In both we get the rapid progressive muscular wasting, paralysis, and cedema. In both the various symptoms of a peripheral neuritis are apparent. Indeed, so like are the two diseases that in reading a description of the alcoholic neuritis one might almost pass it for a reasonably good description of Beri-Beri. I even venture to add that, without knowledge of the patient's surroundings, and uninformed as to the history, an experienced medical man might find it very difficult to differentiate the two diseases at the

bedside of an isolated case of either. The points which would assist one to decide would be these: in Beri-Beri the subject is generally a male; there will probably be no history of alcoholism; his mental condition will be calm and passive; his voice will probably be affected; his heart will not be normal; he will have no tremor and rarely marked tenderness of particular nerve trunks; very little pain, or hyperæsthesia or numbness in the area of any particular nerves. In alcoholic paralysis, on the contrary, the subject is oftener a female, the mental condition is generally that of peculiar restlessness and insomnia; there is a history of drinking habits generally obtainable from the friends, though rarely from the patient; the voice is not often affected, nor is the heart in alcoholism; there is almost invariably marked tremor and pain and hyperæsthesia. On enquiry it will not probably be found, as in the case of Beri-Beri, that there are other cases of the sort in the immediate vicinity. Lastly (of no practical importance for diagnosis, however), the results are very different. In Beri-Beri, according to my experience, the prognosis is as serious as it could well be; in alcoholic paralysis the prognosis is good, and treatment satisfactory.

Sub-acute anterior poliomyelitis has symptoms and progress not very unlike Beri-Beri. The differences are that in Beri-Beri the superficial reflexes are never lost, the sensory symptoms are marked, the muscles of trunk, head and neck are rarely, if ever, affected. Bulbar paralysis never supervenes in Beri-Beri, and febrile symptoms are common, which is just the reverse of what obtains in anterior poliomyelitis. Further, in Beri-Beri the tendency is not towards recovery, the progressive wasting and paralysis does not occur in well-marked groups, there is no tendency to convulsive movements or tremors, and the onset is insidious. In Beri-Beri, commencing invariably with slight weakness in the legs, followed by paresis of flexors of feet and extensors of hands. These, together with the striking tenderness of muscles and early loss of knee-jerks, make the difference more marked than in the previous case (alcoholic paralysis).

Anchylostomiasis has been already mentioned in this paper as a disease which has been mistaken for Beri-Beri. The absence of the ova of the *dochmius duodenale* in Beri-Beri, and the paralysis in the latter disease and the frequent absence of anæmia, are sufficient to distinguish them from one another.

Beri-Beri has been attributed to a scorbutic taint. The sub-cutaneous hæmorrhages and ulcerations of scurvy are absent in Beri-Beri, and anti-scorbutic treatment has, at any rate in my hands, failed to produce the slightest beneficial effect.

The "sleeping-sickness" of Africa has been diagnosed as Beri-Beri, I am told; but, never having seen a case of sleeping-sickness, I am not prepared to venture any further than the remark that, according to the descriptions of this disease, the drowsiness of Beri-Beri is about the only point in common.

In connection with the treatment of Beri-Beri, there are four principal agents which appear to have a curative influence:—

(1) The administration of strychnia, by mouth or intra-muscular injection.

(2) The application of electricity to the affected

muscles, either faradic or galvanic according to circumstances.

(3) A generous, nutritious and nitrogenous diet, combined with a sufficiency of fresh vegetables.

(4) Removal of the patient from the endemic area, which, by the way, could not often be done with the poor unless some generous person would build a convalescent home for cases.

Strychnia is the only drug which has given satisfactory results in my hands. I have tried many others without success. I was even induced to try ergot by a brother practitioner who thought that he had obtained good results with this drug. In my case my patients derived no benefit from its use. Nitroglycerine was tried with no better result, and likewise many others. Of course various remedies are useful for treatment of the various symptoms—*e.g.*, nitrite of amyl for urgent dyspnoea was found useful sometimes.

In the ordinary cases of Beri-Beri under my care in the various estate hospitals my usual directions were to immediately open the bowels with a mild purge. Then to administer thymol, in order to get rid of the *anchylostoma* which is so common in Borneo. After this the strychnia treatment was commenced, the patient taking liq. strychnia B.P. in seven or eight, or more, minim doses three times a day. This (the strychnia) was continued throughout the course of treatment. Symptoms occasionally demanded special treatment; anæmia would be treated with arsenic and iron, malarial conditions with quinine, excess of dropsy with digitalis and diuretics, &c. Surgical measures were sometimes called for—for instance, the removal of accumulated fluids in cavities. I remember even tapping the pericardial cavity in the case of a patient dying in extreme agony. My temerity was not rewarded, however; the patient's life was not even prolonged, and I am not at all certain whether it was not shortened. Anyhow, I never ventured to repeat this heroic (?) treatment. Pilocarpine was found occasionally useful in promoting a healthy action of skin where there was unusual dryness. I used to give $\frac{1}{2}$ gr. hypodermically for several evenings in succession.

Electrical treatment always accompanied the various other measures. It consisted in the daily application of the faradic or galvanic currents to the affected parts. The faradic preferably; but when the muscles had lost their power of reacting to faradisation they were made to respond to the "make" and "break" of the galvanic. The very weakest current only, which would cause a decided reaction, was used. Every morning each patient had a dose of the "battery," and every evening the attendants were supposed to massage the affected parts and well douche them. For the greater convenience of carrying out this treatment I had set apart a separate building for the Beri-Beri cases, so that one could have the electric apparatus always at hand, and moreover, the hospital attendants were thus more easily drilled into their duties by getting into a daily routine of battery and douche for each patient every day.

APPENDIX.

Case of Mr. D. (a Dutchman).—May 6, 1890, aged 33, married.—A strong, well-nourished but spare man,

THE SHARP-TAILED FILARIA OF BRITISH GUIANA.

C. W. Daniels, M.B., Colonial Medical Service, British Guiana, in the *British Medical Journal* of June 17, 1899, describes his observations concerning the probable parental form of the so-called sharp-tailed filaria found in the blood of the aboriginals of British Guiana. Previously, in the same Journal, April 16, 1898, Dr. Daniels described some adult filaria met with in two of these aboriginals. He showed that they were the parental forms of the blunt-tailed blood worms, named *Filaria Ozzardi*, by Dr. Manson. Dr. Manson has added still further to our knowledge of filaria lately, by demonstrating the fact that these adult filaria (*Filaria Ozzardi*) are identical with the parental form of the *Filaria perstans* met with in Africa. Dr. Daniels now believes that he has found the parental form of the sharp-tailed embryos.

Whilst engaged in a *post-mortem* examination of a native of British Guiana, in whose blood both the blunt and sharp-tailed filariæ had been met with, Dr. Daniels found not only a few adult filariæ of the blunt-tailed species, but, in addition, a female, and a portion of a male filaria of a kind new to him. Dr. Daniels infers that these are the parental form of the sharp-tailed embryos. This conclusion seems justifiable, as no other filaria than the two mentioned were met with in the blood of the patient during life.

The two worms closely resembled the *Filaria Bancrofti*. They lay close together in the subperitoneal tissue between the peritoneum and the anterior wall of the abdomen.

The dead worms were arranged in loose coils and did not appear to be encysted or contained in any vessel.

	<i>Filaria Bancrofti</i> .	<i>Filaria</i> No. 1. (<i>Filaria perstans</i> .)	<i>Filaria</i> No. 2. (<i>Filaria Ozzardi</i> .)
	Mm.	Mm.	Mm.
Length	85 to 90	70 to 80	81
Greatest thickness	0.20 to 0.26	0.120	0.210
Diameter of head	0.055	0.070	0.050
Diameter of neck	0.049	0.054	0.039
Distance from head—			
(1) Of vaginal outlet	0.710	0.600	0.710
(2) Of ovarian opening	0.920	?	0.850
Distance from tail of anal papilla	0.225	0.145	0.230
Termination of tail	Blunt, circular, not bulbous.	Slightly bulbous; covered by thickened cuticle prolonged into two triangular appendages.	Bulbous, cuticle not thickened.

A perusal of this table shows that the newly observed filaria is sharply differentiated from the other aboriginal filaria by its smaller head, its large body and the absence of caudal cuticular appendages: on the other hand, its resemblance to *Filaria Bancrofti* is very marked. They are however, not identical. In *Filaria Bancrofti* the body, of fairly uniform thickness throughout, tapers slightly towards the head for a short distance from the vulval aperture, and then more abruptly to the neck. In the newly described filaria, at the junction of the anterior third of the animal with the rest of the body, it attains its present thickness, and from this swelling the body tapers gradually towards the head; the tapering being more rapid for a considerable distance from the vaginal outlet to the neck without any abrupt change. The tail also of the newly observed worm is more bulbous than is the tail of the *Filaria Bancrofti*.

The portion of the male found was about 38 mm. in length. In thickness it was nearly 0.2 mm.; in marked contrast with the 0.11 mm. of the male *Filaria Bancrofti*, or the 0.06 of the male of the other aboriginal filaria. The tail was tightly coiled. There were no protruded spicules; but without running the risk of damaging the specimen, a thorough examination of this feature was impossible. The tip of the tail protruded from the coil. It slowly tapered to a point 0.27 mm. from the tip, where it abruptly tapers. This point probably marks the point of emergence of the spicules. The gradual taper ending in a faintly bulbous tip in this worm is very different from the termination of the tail in *Filaria Bancrofti*.

It is obvious that the powerful body and small head of this worm are better suited for extensive wanderings than the larger head and weaker body of the aboriginal filaria previously found. If its habitat is the connective tissue, as seems probable, it will be very difficult to find; in two previous examinations in which the sharp-tailed embryos had been found Dr. Daniels failed to find the corresponding adult worms. Under the circumstances he considered it advisable to leave the details of structure, and particularly the character of the embryos in the ovarian tubes, for further examination, as they could not be determined without risk of damaging or altering the specimens.

The differences observed both in the male and female are sufficient, Dr. Daniels considers, to differentiate this from the other described adult filariæ; and the name "*Filaria Ozzardi*" might be retained for the new species.

FILARIA BANCROFTI *v.* **NOCTURNA.**



Fig. 1.—2 Males and 1 Female.

FILARIA OZZARDI.



Fig. 2.—Male, Caudal half.

FILARIA PERSTANS.

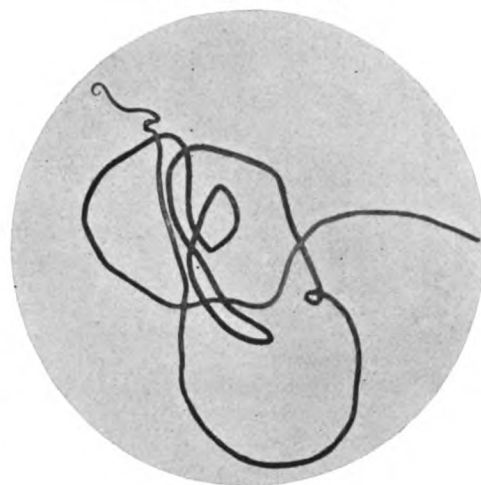


Fig. 3.—Female entire.



Fig. 4.—Head, Male.



Fig. 6.—Head, Female.

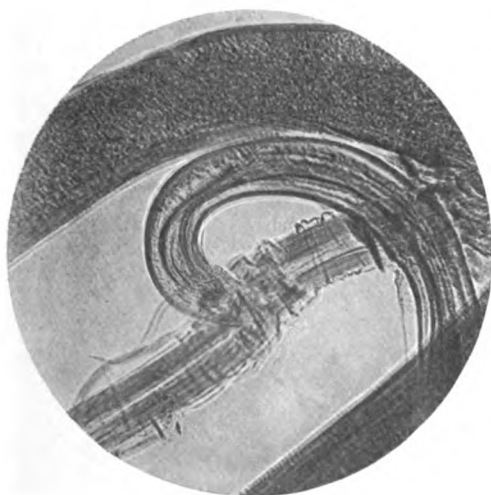


Fig. 7.—Tail, Male.



Fig. 5.—Head, Male.

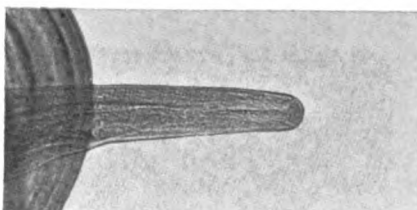


Fig. 8.—Tail, Male.

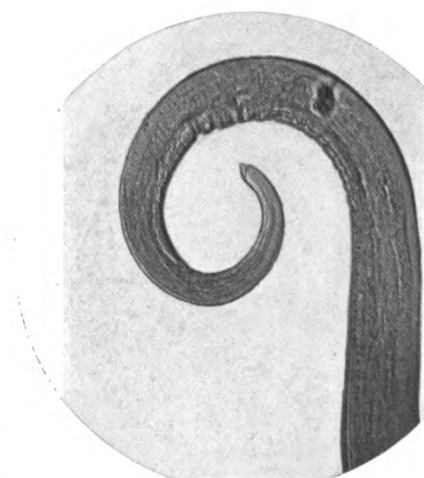


Fig. 9.—Tail, Female.

TO ILLUSTRATE PAPER ON FILARIA BY C. W. DANIELS, M.B.

FIGS. 1, 2, 3 \times 6
FIGS. 4, 5, 6, 7, 8, 9 \times 100

Photo., Francis Fowke, F.R.M.S.]

[Engraved and Printed by Bale & Danielsson, Ltd.

with ruddy complexion. Born and lived in Java before coming here (Borneo) five months ago. Previous health very good. Much hard work lately, and much exposure to weather during rainy season, surveying land and superintending the clearing of jungle for new tobacco-fields. Has been living, he admits, too economically lately, chiefly on rice and dried fish. A moderate drinker, chiefly of gin. Sick six days with malarial fever (intermittent) and diarrhoea, accompanied with swelling and aching pain in lower extremities. Complains of a general feeling of illness.

On examination his general appearance is that of a healthy man. Has soft oedema of feet and legs. Patellar reflex very faint. No tremor. Marked calf and slight general muscular tenderness. Tenderness in popliteal spaces. Gait normal, but weak. Feels as if "walking on wool." No other impairment of sense. Taste normal. Sight good. Sexual power impaired. Urine, S.G. 1020, no albumen, and in every other respect normal. No anæmia. Ordered to stop work, to take absolute rest with medicine:—

R Liq. arsenici hydrochlor. ℥. vj.
 Liq. strychniæ ... ℥. x.
 Aquam ... ad 1 oz.
 M. ft. mist. T.d.s. post cibo.

Diet: No rice; milk, a pint; sago, eggs, *ad lib.*; chicken broth *ad lib.*; beef, 6oz.; cooked vegetables *ad lib.* (in spite of diarrhoea).

May 8.—Patient feels better. No fever. Bowels normal, gait stronger. Continue treatment.

May 10.—Not so well. Bowels costive (!). Very sleepy. Complains much of pain in epigastrium. Voice weak and altered in pitch (a hoarse whisper). No catarrh. Says his hands and feet feel heavy, and "things look dark." Slight oedema of face as well as legs, especially after sleep. Sensation unimpaired. No longer has the sensation as if walking on wool. Patellar reflex absent. Muscular strength impaired, particularly the extensors of feet and hands. Arsenic exchanged for mag. sulph. (a drachm) in mixture. Continue same diet.

May 12.—Feels better to-day. Went out for a walk contrary to my orders. Bowels more regular, and abdominal uneasiness gone. Oedema gone. Pat. reflex faintly present. Looks fresher and better. Fæces examined microscopically. Few ova of anchylostoma discovered.

May 14.—Looks quite well to-day. Complains only of tenderness in flesh and bones. Sight normal now. Pat. reflex marked! Sensations normal.

May 20.—Allowed to return to work, but advised to leave the country.

February, 1892.—I have heard from Mr. Decker. He is in Java, having left Borneo by my advice in 1891. He is strong and well.

Case of Mr. W. (Eurasian Dutch).—July, 1890, aged 28, married. A very short, pale man, but strong and healthy-looking. Born in Java. Always lived there until he came here seven months ago. Previous health good. Much exposed to weather. Working lately in jungle, preparing tobacco fields. Living poorly, occasional debauch. A gin-drinker. Sick three days (preceded by malarial fever), with swelling of legs and feet, pins and needles in extremities, aching of bones. Pain in epigastrium, anorexia, constipation.

On examination the general appearance is normal. No anæmia. Slight oedema of feet and legs. No tremor. Pat. ref. is good. Muscular tenderness is marked. Sensation impaired in lower limbs (no particular areas of partial anæsthesia). Tenderness in popliteal spaces. Gait normal in character, but weak. Taste, sight, hearing normal. Urine is normal. Ordered to rest. (This was on July 22). No medicine.

July 23.—*Tr. stropanthus* with sp. nit. eth., ordered t.d.s., wine and good wholesome diet.

July 24.—Sensation normal. Oedema less. Stomach trouble gone. Pat. ref. has disappeared! Superficial reflexes well marked. Fine "intention-tremor" from general debility, I suppose. Only on movement.

July 25.—Numbness on soles of feet. All oedema gone.

July 29.—Yesterday patient was allowed to take a short walk. To-day oedema has returned, and patient complains of cramps in calf-muscles, and aching pains in hips, knees, and ankle-joints, also pain over liver region. T. 100.4°. Pulse 120. Face oedematous. Feels as if walking on wool.

July 31.—Oedema has increased. Patient feels very ill. Fæces examined show ova of *trichocephalus* and *anchylostoma*. Calomel gr. x to be taken to-night, and mist. sennæ co. to-morrow morning.

August 1.—This morning, after the draught the following was given:—

Thymol, xxv. grs. in black coffee, at 7 o'clock.	
Do. do. do. 9 "	
Do. do. do. 11 "	

August 2.—The thymol had a very powerful action, causing much distress. Many *anchylostomata* found in stools. Ordered liquor strychnia and acid. nit. hyd. dil.

August 4.—Feels much better. Has still a little oedema. Pat. ref. present. Sensation normal. Ordered douche bath every morning.

August 6.—All oedema gone. No more symptoms. Allowed to do half duty in the fields.

August 29.—Patient, who has been apparently quite well since last note, applies again for treatment. Severe abdominal pains. Ravenous appetite. Tertian fever. Has patches of partial anæsthesia on left calf. Pat. ref. good. Gait normal, but weak. Ordered to return to dis strychnia mixture, &c.

A day or two after last note patient by my advice left the country for Java. I have since heard that he entirely recovered from his slight attack of Beri-Beri. He was killed falling off his horse, I hear.

The above are taken straight from my note-book, with all their many imperfections. They were notes scribbled at the bed-side from day to day. Neither cases are typical in the least, and if seen elsewhere than in a region where Beri-Beri raged (the tobacco fields of B.N. Borneo were very hotbeds of the disease in 1890), would probably not have been diagnosed as such.

[Both these gentlemen were "assistants" on the estates of "David Bay (B.) Tobacco Planting Co." P. T. C.]

THE TREATMENT OF DYSENTERY.

BY ARCH. MCKENZIE, M.D. EDIN., M.R.C.S. ENG.

THE treatment of dysentery is often most difficult and anxious, and those who have had any experience in the matter will have come to the conclusion, I think, that, whatever treatment is adopted, we must expect the disease to prove fatal in many cases when it is of a severe type, when it has not been treated from the very beginning of the attack, when the patient is in a low state, and from other causes, or when two or more of these conditions combine in any given case.

In Durban, where I practise, we have a considerable annual experience of dysentery, and we have very early in such circumstances to ask the question, "How can we reduce to its lowest possible degree the death-rate amongst those who become the subjects of the disease in its most severe and dangerous forms?"

My experience in Natal extends over sixteen years and it has led me to the following conclusions, which may, I hope, be of some value to those at least who have to commence practice in tropical or semi-tropical countries where dysentery prevails.

It is most important to commence treatment on the first appearance of the disease. Many patients will treat themselves for four or five days, and by the time skilled advice is called in the disease is too far advanced, the patient's strength too low, and his blood too saturated with the toxins and other poisons absorbed from the bowels to make the chance of success at all likely.

The three lines of treatment likely to be of use are—

- (1) That by ipecacuanha;
- (2) That by some form of castor-oil emulsion; and
- (3) That by sulphate of magnesia or some similar saline, e.g., Glauber's salts.

My experience of ipecacuanha is disappointing. It has often been most difficult to get the patient to retain it. There are many contributors to the literature of this subject who make light of this point. They declare that if you carry out the following well-known instructions upon its administration, viz., give a small dose of opium a little beforehand, ensure that no food, and especially no liquid, be taken for some hours before and for two or three hours after the dose; apply a hot mustard poultice over the epigastric region and make the patient be very quiet and try to resist any tendency to sickness by a strong effort of the will, complete success will follow the treatment. One may readily admit that if you can get the patient to retain two or three doses of about 40 grains of pure pulv. ipecac. in the twenty-four hours, the case will rapidly improve, the pain, tenesmus, mucus, and blood will all give way to a condition of comfort and natural yellow motions, and in this respect all who have studied the matter will admit that ipecacuanha is a most marvellous and efficient drug. My experience is that out of every four or five serious cases of dysentery you will have several in which—apart from the administration of any drugs—vomiting often of a very severe kind is a marked and distressing symptom. If ipecacuanha be

administered to five severe cases it will be found that perhaps two cases retain it and benefit by it, the other three cases will reject it time after time, and, if the greatest discretion and judgment be not exercised, an attempt to persevere with it may be carried too far, valuable time may be lost, the patient may be much reduced and weakened by the attempt to continue the drug, until when it is at last abandoned as hopeless the patient may be similarly summed up.

A large number of the cases of dysentery met with in practice, even when well pronounced, will with careful diet, rest in bed, and any of the well-recognised methods of treatment by drugs, make an excellent recovery, and the importance of deciding upon the best plan of treatment really thus becomes narrowed down to those severe cases which tend to prove fatal, and has for its object the reducing of the fatal cases to a minimum. In my experience it is in the treatment of these cases that one's failures by ipecacuanha leave one with a patient whose attack is sufficiently severe to tend strongly to a fatal issue, and whose condition has been rendered worse in the unsuccessful attempt to treat by this method, during which valuable time and the patient's strength have been largely drawn upon, or even as the event may prove, over-drawn upon.

The second mode of treatment by castor oil is useful. Like all methods it is best preceded by a mild dose of 3 or 4 grains of calomel, and the sooner treatment is commenced the better. I generally prescribe something like the following:—

R	Ol. ricini opt.	℥ lxxx.
	Liq. sodæ	℥ ii.
	Sod. salicyl.	gr. v.
	Glycerini	℥ xlviij.
	Mucilaginis	q. s.
	Aq. menth. pip.	ad. 3ss.

Sig. 3 ij. every hour, or 3ss. every two hours.

A dessert-spoonful of this may be given every hour, or at first oftener, till the motions improve, and then a little less frequently each day until quite healthy motions are established.

If there is much pain and straining, 1 or 2 minims of tinct. opii may be given with each dose, or a few grains of pulv. ipecac. co. every three or four hours till these symptoms become abated. The bowel may be freely washed out by a copious douche of sterilised boracic acid lotion three or four times a day, a soft rubber catheter being gently floated up the rectum and sigmoid for this purpose.

The third method of treatment is that by salines. The sulphate of magnesium, or of sodium, is the salt generally selected. For the last six years or so I have followed this line of treatment, and can add my testimony very strongly to that of those who have drawn attention to its value. The following is the prescription I have fallen into the habit of using:—

R	Magnes. sulphat.	Si.
	Quin. sulphat.	gr. iss.
	Acid. sulph. aromat.	℥ viij.
	Hydrarg. perchlor.	gr. ʒv.
	Glycerini	3iss.
	Inf. quassia	ad. 3 ss.

Sig. Give a table-spoonful every hour.

This mixture may be made up in twelve-ounce bottles and should be pushed at first. The first four

to six doses should be given every fifteen or twenty minutes. The motions soon begin to show the effects of the medicine. The slime, blood, and straining give way to pale, rice-watery-looking motions without straining. This usually occurs in eight to twelve hours. There is soon after this a yellowish colour added to the motion, and in twenty-four to forty-eight hours this improved colour is thoroughly established. The frequency at first of the motions is not any less, but the painful straining usually disappears as if by magic, and when the abdominal pain is very great from the first, or accompanied by painful tenesmus, it will be found very useful to give a few drops of laudanum every hour or so along with the mixture until the good effects begin to establish themselves. The mixture must be given less often when the motions have become decidedly yellow, and then it will be found that the bowels cease to act so frequently, and by the third or fourth day one usually has about one motion every three hours, and from this condition the patient steadily progresses to recovery. It is a mistake to be sparing in the use of the mixture at first, and it is unwise to stop it or decrease the dose too soon. The most important guide in this matter is the colour of the motions, and until a good yellow colour is established no real improvement can be relied on. The quinine, mercury and sulphuric acid contribute their valuable therapeutic effects, and in cases in which there is a malarial infection complicating the dysentery, the quinine is of especial value.

PRICKLY HEAT.

By ST. GEO. GRAY, M.B., B.CH. UNIV. DUBLIN,
Colonial Assistant Surgeon, Castries, St. Lucia, West Indies.

PRICKLY heat is an affection which is seldom brought to the notice of the physician, being generally considered a minor ill, not affecting the general health, leaving no permanent results, and disappearing spontaneously as soon as its cause is removed.

The annoyance that it causes is only temporary, and as every new arrival in the tropics expects to suffer from it more or less, few of the sufferers think it worth while to consult their doctors about it. Mr. Frederick Pearse, of Bombay, has written an excellent "Note on Prickly Heat" in the JOURNAL for June, but it is not quite clear upon what grounds he bases his opinion that it is a seborrhœa and not a form of miliaria (misprinted *malaria*). If prickly heat is a seborrhœa, how is it that it is seldom or never seen in the scalp, where seborrhœa is by no means uncommon? In seborrhœa there is an increase of the sebaceous matter, but he says, with reference to treatment: "It is not sufficiently borne in mind that soap removes sebaceous matter from the surface of the skin; that the skin thus freed from its natural oil tends to become dry, rough, and hard; that the sebaceous glands are thus unduly stimulated to produce more secretion, while at the same time the excessive perspiration is also irritating them to lubricate the surface. The removal of the natural grease of the skin under these circumstances is distinctly disadvantageous, and bathing should be performed with plain water."

I entirely concur in the greater part of this statement, but I maintain that the sebaceous glands are not *unduly* stimulated to produce more secretion, but that the removal of the natural oils from the skin renders it more susceptible to the influence of moisture, i.e., excessive perspiration, and so predisposes it to prickly heat.

S. Pollitzer (*Journal of Cutaneous and Genito-Urinary Diseases*, February, 1893) has carefully studied the histology of prickly heat and shows that the vesicles are retention cysts caused by the stratum corneum becoming sodden by being soaked in perspiration. The cells of the horny layer imbibe the water and swell so as to occlude the orifices of the ducts of the sweat glands. "The stratum corneum," he says, "was almost everywhere thickened, not however, through increase in the number of cells, but on account of the enlargement of the individual cells. . . . The uppermost layers of the stratum corneum were frequently lifted up by fluid, constituting a vesicle usually with clear contents. These vesicles appeared also to be connected with sweat ducts; certainly a sweat duct could often be seen in line with the vesicle. . . . The most striking change found in prickly heat is the cystic dilatation of the sweat ducts. It is this change which constitutes the chief clinical feature—the minute discrete vesicles. . . . This condition is evidently due to imbibition with water, with sweat. An epidermis which is bathed in perspiration, retained as it commonly is by wet underwear, cannot properly cornify; and in imbibing water the cells swell, and swell of course in all directions, laterally as well as vertically. The line of cells, therefore, expanding may easily be pushed over the orifices of the ducts occluding them at a time when the secretion of sweat is momentarily in abeyance." Here, then, we have an indication for prophylaxis. Where the perspiration evaporates rapidly or can be drained away by absorbent underwear there is little or no prickly heat. And clinical experience bears this out. As the sufferers generally look upon it as an inevitable accompaniment of their residence in the tropics, and as it is also looked upon by many as a "healthy sign," it is rarely brought to the notice of their medical attendant. Most of my investigations, therefore, have been made on my own person. When I first came to the tropics I wore the thinnest of cotton underclothing and was hardly ever free from prickly heat all over my body. When wet with perspiration this was extremely uncomfortable, and I soon realised that cotton was not the ideal material for underclothing in the tropics. I then began to wear wool next to my skin, with the result that prickly heat has never troubled me since, except on those parts of the body not covered by wool or exposed to the air, viz., the forearms *from the lower edge of the woollen sleeve of the undershirt to the lower edge of the linen cuff at the wrist*. Several of my patients who wear woollen underclothing have prickly heat only on their forearms.

The wool absorbs the perspiration as fast as it is secreted, and carries it away from the skin to the outer garments, so that the epidermis never becomes sodden, and consequently the orifices of the sweat ducts remain patulous.

Many people object to wear wool in the tropics, but it need not be heavy, the thinnest material will do provided it is wool. Woollen underclothing is also the best protective against the sudden changes of temperature which are so common in the tropics, and is certainly more comfortable, even in the hottest weather, than wet cotton or linen next to the skin.

As to bathing, by all means let the subject of prickly heat bathe in plain, cool water, without soap. In such a case there is nothing more grateful than a good soak in a tub of rain water, or better still, in a mountain torrent, until thoroughly cooled—not chilled—then a brisk rubbing with a rough towel, and afterwards gentle exercise to prevent a chill, but not sufficient to cause undue perspiration.

As to curative treatment, Mr. Pearse says, "Lanoline when freely used (and it must be very freely used), and when combined with a pleasant oil, is the most successful application." His very treatment, which is undoubtedly the correct one, shows that he perceives that there is a deficiency and not an excess of sebaceous matter in the skin, which would be the case were prickly heat a seborrhœa.

Theoretically and practically, I have no doubt that the free use of lanoline is the very best curative treatment for prickly heat (always excepting change to a cooler climate), but I do not think that he will find many Europeans who will submit to it for any considerable length of time. The thorough anointing of the entire body twice a day with lanoline "combined with a pleasant oil" can only be undertaken by those who are either able to pay a big laundry bill (for sheets and underclothing could be used only once) or who are willing to live in the state of those negroes who delight in rubbing themselves with tallow (soft candle, *chandelle molle*) and cocoa-nut oil. In the latter case it is better for them to retire into private life.

The only certain and permanent cure for prickly heat is cold weather. On removal to a cooler climate the eruption disappears as if by magic, however intractable it may have been before, only to appear again with all its attendant discomfort when the sufferer returns to the tropics.

MALARIAL HÆMORRHAGE.

The *Revista Med. de Bogota* contains four observations related by P. Q. Romero, of Barranquilla, in which severe hæmorrhage subsided at once upon the administration of quinine. Each case deceptively simulated another affection. In one, there was profuse metrorrhagia, fever, and debility, suggesting acute metritis, but the patient recovered at once with quinine, and another attack, a few months later, yielded to it in the same way. The second case simulated pneumonia, with fever and blood-streaked sputa, but all the symptoms subsided with quinine. The third case, with excessive hæmoptysis, fever, night sweats, cough, and emaciation was supposed to be tuberculosis. Quinine arrested the entire syndrome, and by the end of the week the patient was entirely restored to health, with no recurrence of any of the symptoms to date. The fourth observation was a case of sudden, severe, rebellious epistaxis, which he treated with quinine instead of tamponing, with almost immediate success. He always combines a few brief cold baths with the quinine treatment.—*Medical Brief, May*.

THE RELATION OF THE SOLDIER'S DRESS TO HEAT APOPLEXY.

Lieutenant-Colonel McCartie, of the Indian Medical Staff, contributes to the *Indian Medical Gazette* for June a most interesting communication on "The Cause and Prevention of Heat Apoplexy in the Army," and one which will have special force for us in regard to our troops now serving in the tropics.

Colonel McCartie first combats, and we think satisfactorily, Dr. Sambon's views, published over a year ago in the *British Medical Journal*, that "heat apoplexy" is not caused by heat, but is an infective disease. He points out that soldiers and civilians work under such different conditions as to dress as to account fully for the prevalence of the disease among soldiers, while civilians working under a higher temperature are almost exempt. The latter wear a thin twill cotton shirt, light, loose cotton trousers, cool socks, cool light shoes, a light gossamer coat, worn open, and a thick pith hat, coming down the neck, and carry a thick umbrella; while the soldier wears clothing unnecessarily warm and so buttoned up and strapped down that the air cannot circulate about his body, the evaporation of the perspiration cannot go on, and thus Nature's heat-regulating mechanism is thrown out of gear. The consequences are distress, fatigue, exhaustion, fever and heat apoplexy!

Colonel McCartie's observations are based upon considerable experience, especially during the hot weather campaigns of 1895 and 1897. He noticed that most of the men attacked by heat apoplexy were very warmly clad, most of them wearing flannel shirts, and many even thick woollen undershirts as well.

One piece of evidence recorded is very striking. In a frontier campaign in 1897 sufficient coolies could not be obtained, and part of the baggage had to be carried by native soldiers in plain clothes. Before the end of the march, says Colonel McCartie, nearly all the men in the ranks were utterly exhausted, and some of them had heat apoplexy, while their comrades doing duty as bearers and carrying a much heavier weight, but dressed in their own loose, light, rational dress, were not the least distressed.

Colonel McCartie condemns the belts and straps which go over the coat as the worst part of the soldier's dress, and advises doing away with them all. He recommends a loose Norfolk jacket with four pockets, loose trousers with two pockets in front and two behind, and a loose twill cotton shirt with two pockets. To these might, if necessary, be added a light knapsack capable of carrying the contents of the haversack and the greater part of the ammunition. A hook could be fastened in the jacket for the water bottle, and a frog attached for the bayonet.

The accommodation afforded by the pockets would do away with the necessity for belts, straps and pouches; while on the march the knapsack would accommodate all the ammunition and food needed.

To sum up, the rational attire for soldiers serving in tropical climates would be light, loosely made garments of very porous material, and an entire absence of straps, belts, pouches, &c. The soldier is not dressed in parade order for fatigue duty, and campaigning is fatigue duty in the highest degree.—*New York Medical Journal*, July 22.

VACCINATION AGAINST YELLOW FEVER.

M. Domingos Freire, Director of the Bacteriological Institute at Rio, writes in support of his micrococcus, which he considers pathogenic of yellow fever, Sanarelli's bacillus being merely an agent of secondary septicæmia. His strongest argument is the efficacy of vaccination with attenuated cultures of the micrococcus in question. Between 1888 and 1897, 12,665 such vaccinations were made with the result that, while the mortality of those not inoculated reached 80, 40, and even 50 per cent. the mortality among the inoculated varied between 0.8 and 1 per cent.—(*Revue Scientifique*, No. 10.)—*Janus*, May.

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THE

Journal of Tropical Medicine

AUGUST, 1899.

DR. THIN'S PRESIDENTIAL ADDRESS AT THE SECTION OF TROPICAL DISEASES.

DR. THIN's presidential address at the opening of the section of Tropical Diseases may be divided into two parts: one relating to the scientific problems connected with malaria, more especially with the rôle of the mosquito in the causation of the disease; the other concerning the schools in England in which tropical medicine should be taught. In the first part, a very succinct account is given of the work of the Italians, formed on the lines of investigation initiated and brought to such brilliant results by Manson and Ross. The experiments of Bignami, Grasse and Bastianelli on the development of the malarial parasite in the body of *Anopheles* were given, and the provisional conclusions to be deduced from these observations and experiments:—viz., that the larvæ of *Anopheles* do not carry infection; that it is only those *Anopheles* which have bitten human beings suffering from malaria that were capable of propagating the disease; that the yellow brown spores found in *Anopheles* infected by human malaria are equivalent to the black spores described by Ross in the grey mosquitoes which have bitten birds infected with proteosoma. And

instead of being the first stage of a phase of existence of the hæmatozoon, which has not yet been discovered, it is possible that they are degenerating forms. It is evident, however, that the Italian observers have made no special advance on the information already given by the experiments of Ross.

It would have been well if Dr. Thin had been satisfied with following the beaten tracts of science, and had not diverged into the interesting but doubtful paths of controversy, for he was not particularly felicitous in his handling of the delicate and somewhat needlessly volcanic question connected with the teaching of tropical medicine in England. Controversial subjects are usually best left alone in Presidential addresses, unless it is known beforehand that the sympathy of the majority is sure to be on the side of the speaker, and, even then, it is questionable whether it is wise, because they create in a portion of the audience a certain amount of opposition to the Chairman, which is best avoided. There are plenty of opportunities of raising questions which may seem of importance to those who hold strong views upon them, by reading a paper or opening a discussion on the subject, and then opinions may be ventilated in the freest manner without in any way infringing the canons which are usually considered to belong to Presidential addresses.

We sympathise with Dr. Thin in the anomalous position he was placed by having the motion on his address defeated and an amendment carried. The probability, or even the possibility, of such a contingency should have been foreseen and weighed, for, if the march of events during the past two years had been closely examined and judged, it required no great acumen to prognosticate the result, and thus a disagreeable incident would never have happened. Viewing Dr. Thin's remarks in the most charitable light, there is no doubt that they placed him in the hands of his opponents, for, however adroitly introduced by him, they were manifestly an attack on the Liverpool and London Schools of Tropical Medicine, and more particularly on the London School. Why the President of the Tropical

Section should have seen fit to endeavour to throw the apple of discord into the new Tropical Section of the British Medical Association is scarcely worth while considering. The comparative powerlessness of the profession in proportion to its numbers and the position and education of its members has been attributed to the internal dissensions and jealousies to which it is said to be a prey. We have ever been averse to this opinion, but incidents do occur now and again which seem to favour the contention. As a matter of fact, Dr. Thin's proposals were discussed before either the Liverpool or London Schools of Medicine were created, and it was found that nothing short of the establishment of these schools could meet the advancing requirements of the Colonies. None doubt the excellency of the Netley School, and the good work it has done and is doing for the Army Medical Services, British and Indian. Its good name is safe in the hands of these services, and long may it flourish, giving to its cadets a special training, not only in military medicine, surgery and hygiene, adapted for whatever part of the world the Army may be called to, but also in that discipline and *esprit de corps* which are so essential for a young officer to obtain at the commencement of his career. To set off Netley against London and Liverpool in the movement for teaching Tropical Disease to medical men likely to be engaged in civil practice in the Colonies or in the Colonial services, is to display misapprehension and confusion as to the purposes for which Netley was established and the objects for which the London and Liverpool Schools have come into existence. The Netley authorities, as a body, are not likely to support any ill-considered scheme that might introduce representatives of other bodies on to the hospital and professional staff of the Netley School, which would possibly endanger or destroy its special character and its value as a military school. However much disposed others might be to risk such a contingency, it is certain that the London and Liverpool Schools would be all the same the centres of attraction, and the forces which have established them are quite competent to maintain them.

EXAMINATION OF STAINED SPECIMENS OF BLOOD.

DR. H. F. HEWES, in an introductory lecture to the Course in Hæmatology at the Harvard Medical School, draws attention to the importance of the examination of stained specimens of blood in its application to clinical work. The failure of many to acquire this experience is attributed to an exaggerated idea of the complexity of the methods involved in each examination, whereas, as a matter of fact, a large percentage of all the information we can obtain from the blood can be secured by a single method, and one so simple in its technique that it can be applied by any practitioner who owns a microscope. This method is the now well-known one of examination of a stained specimen of blood, and may be applied to the disclosure of the existence of an anæmia, a leukemia, or other form of leucocytosis, or to the discovery of the presence of blood parasites, such as the *Plasmodium malarie*, the *Filarie sanguinis hominis* and the spirillum of relapsing fever. A few clean cover-slips and a needle are all that is necessary at the bedside. The blood is spread on the cover-slips and dried in the air. Later, it may be fixed by heating the cover-slips at 110° C. for ten minutes or by placing in absolute alcohol. It is then subjected for four minutes to an Ehrlich three-coloured mixture prepared according to the following formula:—

Ehrlich-Biondi-Heidenhain three-coloured mixture 1·7 grammes.

Acid. fuchsin	0·05	„
Absolute alcohol	2	cc.
Distilled waters	18	cc.

The specimen is then washed in water and subjected for from one half-second to ten seconds to Löffler's solution of methylene blue. It is again washed and then dried and mounted in balsam. Microscopical examination in the majority of cases, Dr. Hewes states, will serve to definitely rule in or rule out the existence of a pathological condition of the blood, and to determine the nature of this condition. If pathological conditions are found about which more definite knowledge is desired, or if any doubt

remains in regard to the blood in the case, the examination may be reinforced or continued by the application of other methods, such as the blood counting and the hæmoglobin estimation. Otherwise we may rest satisfied on the finding of the stained specimen.

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

I.

SCARLET FEVER IN INDIA.

THE diminution of prevalence of scarlet fever as the Equator is approached from north and south seems to be a solid fact. In preparing a paper on the subject some years ago for the Sanitary Congress at Budapest, I tabulated the references to this exanthem contained in Davidson's "Geographical Pathology," and found full confirmation of the rarity or absence of the disease in tropical countries. The information obtainable is not very exact, but there is no reason to dispute its general accuracy, and it is to be hoped that the article published in the last number of this Journal will elicit more precise data. As regards India, a considerable mass of knowledge has been contributed to the *Indian Medical Gazette* since the year 1870. Up to that time it was believed that, as stated in your article, the disease was unknown in India; but there is reason now to believe:—

(1) That scarlet fever has been frequently imported by troop ships, and that small epidemics have thus arisen in Indian natives, especially among children.

(2) That these epidemics have always been of a limited kind and have speedily died out.

(3) That they appear to be more substantial and protracted in hill stations than on the plains.

(4) That cases and groups of cases, have been observed, the origin of which could not be ascertained.

(5) That the disease does not appear to prevail epidemically among indigenous races and populations.

(6) That cases of an exanthem closely resembling, if not identical with, scarlet fever, have been observed in Calcutta and elsewhere. These have been mostly single or limited to one family, and have manifested no disposition to diffusion.

The statistics published by the Sanitary Commissioner with the Government of India, give the following totals for the twenty years, 1877-96 inclusive:—European troops, 56 cases, 2 deaths; women, 7 cases, no deaths; children, 124 cases, 10 deaths; native troops, 20 cases, 1 death; prisoners, no cases. The strength of these sections of population may be roughly stated as 60,000, 4,000, 7,000, 125,000 and 110,000. The figures indicate the immunity of natives and comparative freedom of Europeans in India. In the absence of descriptive detail—clinical and epidemiological—they must be accepted with the proverbial grain of salt: but the communications which have from time to time been published in the *Indian Medical Gazette* possess a higher value and they go to support the conclusion above stated. As this periodical is not generally accessible in this country, I conceive that I shall fulfil a useful purpose by presenting a short precis of the papers which have appeared in it on the subject from the year 1870 onwards.

1870. P. 150, case of erythema scarlatini-forme in a Eurasian girl aged 7, at Saharunpore, by Dr. A. Garden; p. 251, case of scarlatina in the Simla hills (Captain E.), mention made of several similar cases in the same station in children, "well-marked but did not prove contagious," by assistant-surgeon T. Maunsell, R.A.

1871. P. 79, letter from Dr. W. T. Dickson, suggesting that these were cases of rōtheln; p. 124, leading article, drawing attention to the subject and contending that scarlatina is unknown in India; p. 156, details of thirteen cases among the

children of H.M.'s 12th Queen's Royal Regiment at Poona, in May, June, and July, 1870, by assistant-surgeon G. J. Gibson, M.D., origin of outbreak unknown; p. 159, letter from Dr. A. F. Bradshaw, showing that in the years 1860-68, fifteen cases with four deaths had been returned as scarlet fever among English troops in the Bengal Presidency, stating that he had seen undoubted cases in a family at Simla, contending that Drs. Garden and Maunsell's cases were scarlatina, giving details of his own cases and referring to others, mentioning that scarlet fever broke out on a troop ship conveying detachments, and subsequently at Jullundur and Sealkot after their arrival, and hinting at the occurrence of the disease in Calcutta hospitals; p. 169, editorial, acknowledging that Drs. Gibson and Bradshaw's cases were scarlatina; p. 177, account of an outbreak among children of 58th Regiment at Sealkot, following arrival of a draft, by surgeon E. H. Smedy, M.D.; p. 211, case of scarlatina maligna in a private of the 96th Regiment at Dinapore (surgeon H. Mitchell refers to another case at Dum-Dum in 1870); p. 216, editorial, embodying reports by Calcutta physicians. Conclusion stated that scarlet fever does occur in India, in European communities, either as an isolated case or a limited outbreak. It does not appear to show any great tendency to spread and in all cases there is a demonstration or a strong suspicion of its importation into this country from Europe. There is not a jot of evidence that the disease is indigenous or has ever occurred among natives of India; p. 219, letter from Dr. A. Garden, pointing out that his case was isolated in a large family, and doubting the alleged prevalence of scarlet fever among hill natives; p. 237, case of scarlatina in a puerperal woman at Dacca, by Dr. H. C. Catcliffe; p. 267, letter from Dr. C. M. Jessop, contending for the possible origin *de novo* of scarlet fever in India.

1872. P. 43, notice of the Jumna troop ship having been placed in quarantine at Bombay on account of scarlatina and measles; p. 104, article by Dr. A. Garden, admitting that the limited prevalence of scarlet fever among Europeans in India has been established, suggesting that this is

due to the shorter voyage from Europe, and giving details of four cases in a family of five children at Saharunpore, where the origin of the disease could not be traced.

1873. Editorial, stating that in 1871, 110 admissions and seventeen deaths from scarlet fever had occurred among the children of European soldiers in India, and quoting details of an outbreak in the 44th Regiment.

1876. P. 119, notes of three cases treated in the Presidency General Hospital, Calcutta, by Dr. R. D. Murray.

1879. P. 85, case in a Eurasian boy in Calcutta, by Dr. H. Cayley.

1881. P. 177, report of cases in K.R.I. Hussars, by surgeon-major T. Rudd, M.D., origin of outbreak unknown.

1882. P. 50, case in Calcutta by Dr. Juggobundoo Bose.

1883. P. 73, case at Bangalore, by surgeon J. Hoey, origin unknown.

1887. Isolated case observed by Dr. K. Macleod in Calcutta.

1889. P. 151, paper read at the Calcutta Medical Society by Dr. Kailar Chunder Bose, detailing a number of cases of scarlet fever in children, which he had met with in 1886, which had been associated with defective drainage.

KENNETH MACLEOD.

August 9, 1889.

II.

In your "Article for Discussion" in the Journal for July attention is called to the absence, or, at all events, the great rarity of scarlet fever among native and European residents in the tropics, and your readers are asked to send their experience as regards this disease. As one engaged in extensive practice for thirty-two years in Kingston, Jamaica (lat. 17° 57' N.), I would ask permission to make the following observations on the subject.

Scarlet fever I but rarely met with—in fact, I doubt if I had fifteen cases of it in thirty-two years. Unfortunately I have kept no notes, but

I remember that I had but two groups of cases of three or four each, and one of them was distinctly traced to a letter received by mail packet from a family in England among whom the disease was prevailing. The other cases were sporadic, and there was no extension of the disease from any of them. Such was my personal experience. But in former days scarlet fever seems to have been much more common in Jamaica. In the "Report of the Central Board of Health of Jamaica" for 1852, the following observations occur:—"Scarlet fever occurs occasionally in this island, both in a sporadic and epidemic form. . . . In May, 1841, the first case of an epidemic appeared in Spanish Town; from thence it soon spread over the whole island, and raged with great severity and mortality among all classes. Since this, occasional and sporadic cases are stated to have occurred, especially in Kingston, and some of them very lately."

As you correctly state, "the absence of a disease in any country is a scientific fact of much interest." I may add that abscess of the liver, so common in the East, is very rare in Jamaica. During my long residence I only met with three cases of it; one in a European, which discharged through the bowel, recovered; two other cases in natives, on whom I operated, with the result that one died and one recovered.

Perhaps with your kind permission, I may on a future occasion refer to other diseases which I but rarely met with in Jamaica.

IZETT ANDERSON, M.D. Edin.

Eastbourne.

III.

MEASLES IN THE TROPICS.

IN the July number of the JOURNAL OF TROPICAL MEDICINE Mr. Cantlie draws attention to the "Rarity of some of the Common Diseases of Temperate Climates in the Tropics." His paper deals with scarlet fever. I have had, while acting as ship surgeon, the opportunity of observing three cases of measles occurring in the tropics.

Although the cases are few in number, the recording of them may be the means of eliciting from those who have more extended experience

in the study of the subject some valuable information.

May 17, 1895, at Port Said, we took Lieutenant P., his wife, nurse and three children as passengers on board s.s. *City of Dundee*. They had come direct from Constantinople. While speaking to Mrs. P. on May 19, I noticed the baby, about six months old, had a very suspicious-looking rash on the face and arms, and drew the mother's attention to the fact. She said it was "prickly heat," but at my request the nurse undressed the child, whose body was then seen to be covered with a typical rash. Temperature in groin 101° F., cough, running from the nose; the other symptoms of the disease were also well marked. The child got quite well in four days, and neither of the other children were infected.

On March 27, 1897, on board s.s. *Obidense*, of the Red Cross Line, I was told that one of the Portuguese steerage passengers was ill and required to see me. I proceeded to the quarters and found a boy, whom we had taken on board at Lisbon on March 23, was suffering from an attack of measles. I immediately had him isolated. The next day another boy developed the disease, who was also isolated. No other case occurred. Both boys were quite recovered in five days.

Remarks.—The rapidity of recovery in the three cases is very striking, also the slight degree of infectiveness. In the first case the other two children slept in the same cabin as the patient and did not contract the disease, although unprotected by a previous attack. This I put down to the fact that on board ship in the tropics the port holes are kept open night and day, thus diluting, by a continuous current of fresh air, the contagion of the disease. The surrounding temperature must also be a factor. This case was not given any medicine, and spent the day on deck like his brother and sister.

The other cases were quite different in regard to ventilation and hygiene, as anyone with experience of Portuguese and Brazilian steerage passengers will readily understand; they seem to think that fresh air, soap and water, are to be strictly avoided. The bedding (both boys had slept together), clothes, &c., were disinfected and

exposed to light and air. The port-holes of the steerage I got the ship's carpenter to fasten open with stout wire, so that they could not be closed (we were in fine weather, running from Madeira to Para). When we consider that the steerage was full, including some twenty children, it is remarkable that no other case occurred.

W. G. TOTTENHAM POSNETT.

Reprints.

ON TRANSMISSION OF PROTEOSOMA TO BIRDS BY THE MOSQUITO: A REPORT TO THE MALARIA COMMITTEE OF THE ROYAL SOCIETY.

By Dr. C. W. DANIELS.

From the *Proceedings of the Royal Society*, vol. 64.

(Continued from page 339.)

I incised the stomach of infected mosquitoes, and by repeated washing and compression with a cover-glass was able not only to wash out the contents, but even to express the loosely attached epithelium, so as to leave the stomach a transparent, clear bag. The majority of coccidia remained fixed to the outer wall, though in one of the mosquitoes I observed a few coccidia escape with the epithelium. On subsequent attempts to detach the coccidia by this process I failed to do so, though some coccidia would be ruptured.

The next morning the smallest coccidia measured 10 μ , some were 12 μ . On the sixth day they were met with up to 30 μ ; by this time the pigment had absolutely as well as relatively diminished.

In another three days some of them reached 60 μ , and in the last of the series (tenth day) there were coccidia measuring 70 μ .

The coccidia could now be seen to project from the outer wall of the stomach; very few contained pigment, and that only in small amount.

Some of the coccidia were clear, and others had a granular appearance, but in none were either black spores or germinal threads to be seen.

(9) For the observation of the further development of the coccidium, the early deaths of the mosquitoes, owing to the inclemency of the weather, rendered this series useless.

One of the insects infected on the night of January 5, and another infected on January 5, did reach this more advanced stage, and in the last of those fed on January 5, and which died on January 22, ruptured cysts, as well as numerous cysts containing mature germinal threads, were found by me in the stomach wall; these threads were also found in the body fluids and in cells in the salivary glands. In one of the mosquitoes infected on January 5, which died on January 19, the coccidia had an appearance of striation.

In consequence of the effects of the unfavourable climatic conditions on the experimental insects, my observations on the development of the proteosomal

coccidium were mainly made on mosquitoes infected November 30 and subsequent dates before my arrival, and on some infected on December 22.

On adding salt solution (15 grs. to the ounce) to an ordinary slide containing an infected mosquito stomach, and pressing on the cover-glass, a projecting coccidium was ruptured; the contents poured out into the fluid, leaving the cyst wall still attached to the stomach.

The contents were seen to consist of a mass of shrivelled threads. This appearance I frequently observed in the other series of infected insects already mentioned.

These threads, Ross's germinal threads, are sickle-shaped bodies, about 14 or 15 μ in length. They stain with logwood or methyl blue, but not strongly. On adding water or Farrant's solution they lose their shrivelled appearance, and become more rounded. Nearer one end than the other is an unstained portion (? nucleus). They show no signs of movement, but as they are invisible in water, and only become visible when shrivelled by the salt or stained, it may be doubted if they have been seen alive.

If the thorax of the mosquito at a somewhat more advanced stage in the development of the proteosomal coccidium is incised, similar threads will be found in the fluid exuded if salt solution is added. In this case ruptured cysts can be found in the stomach wall.

The relation of the infection to the veneno-salivary gland involves a difficulty not met with in any other part of the examination.

The dissection of the stomach is easy, that of the salivary gland in its entirety is not, and for some reason appears to be more difficult in the old infected mosquitoes. Any rough manipulation results in the detachment of the cells, and little more than the duct is left. In most cases, however, even in old infected mosquitoes, one entire gland, or portions of both, can be exposed in fair condition.

In every case where this was done and in which germinal threads were found in the body fluids, the germinal threads were also found in some of the cells of the salivary gland. I failed to find similar threads in the large number of salivary glands obtained from uninfected mosquitoes bred from larvæ, or caught about the laboratory or from mosquitoes at the earlier stages of proteosomal infection.

The affected cells, as they have a granular appearance, can be distinguished with a low power; the unaffected cells are quite clear.

With a high power, if not very numerous, the isolated germinal threads can be clearly distinguished in the cells; they are recognised by their peculiar shape and shrivelled appearance (the examination must be made in salt solution). If numerous, the individual threads can be better made out in the cells of the salivary gland than in the coccidia of the stomach wall; but, as in the case of the latter, pressure on the cover-glass will rupture the cell, and the germinal threads are then poured out.

The threads do not fill the cell. There is a faintly granular crescentic portion on the side most remote from the duct which, in many cases at least, is free from threads. The part of the cell in which the

threads lie must be nearly fluid, as it permits oscillation of the threads to take place.

The whole of the veneno-salivary gland is never involved. In one dissection made by Ross the cells in both middle lobes and in no other part of the gland contained the threads. In several instances, where one gland has been exposed entire, the middle lobe alone has been involved; but in the majority all that can be stated with certainty is that the cells in one portion of the gland contain threads, and that those in other portions do not.

On these points I have satisfied myself by repeated examination, though the appearances are by no means difficult to make out.

I have gone at some length into the description of this matter, as, so far, we have found no satisfactory method of making permanent preparations. All the preservatives at our disposal, with the exception to some extent of weak formalin solution, wrinkle up the delicate cells; and I have no confidence in this agent as a means of making permanent specimens.

The following specific observations made by myself on mosquitoes dissected by Major Ross, Dr. Rivenberg, of the American Mission, who is working with Dr. Ross and myself, may be of interest:—

(a) Coccidial cysts full of apparently mature germinal threads; no ruptured cysts; no germinal threads in the body-fluids or salivary glands. Two observations.

(b) Cysts full of germinal threads; other ruptured empty cysts; germinal threads in body-fluids; germinal threads in salivary glands. Over twenty observations.

(c) Empty cysts in stomach wall; germinal threads in body-fluids of thorax; germinal threads in salivary glands; no cysts still containing germinal threads. Two observations.

(d) Empty cysts only in stomach wall; no germinal threads in body cavity; no germinal threads in well exposed salivary glands. One observation; the mosquito had been infected four weeks before death.

These observations fully confirm Ross's statement in every point. They indicate that the threads are formed in the coccidia; and that the germinal threads escape into the body cavity on the rupture of the coccidia, to be again collected in the salivary glands.

I should have liked to extend the series, but the continued cold weather renders it improbable that I shall be able to do so before I leave.

10. The infection of birds free from proteosoma by the bites of mosquitoes.

On December 20, the day before my arrival, twenty-two birds were examined and found free from proteosoma. On that night some of these birds were used for feeding the mosquitoes which had been infected on November 30 (?) and on the 24th and subsequent days; the remainder of the birds were used for feeding the mosquitoes first infected on November 30, and December 10, 12, and 15. In other mosquitoes of this series germinal threads were found in the salivary glands; and those which fed, when examined later, gave the results indicated in paragraph 9.

On December 30, Dr. Rivenberg and myself examined these birds; three of them had proteosoma, two in large numbers.

On January 4, I examined them all except one which died on January 2; in this bird the heart's blood contained no proteosomata, and the organs were free from pigment.

Five more of them had now proteosoma; in every instance the parasites were very numerous. On January 6 and 7, I again examined them; three more had proteosoma, also in large numbers.

On January 9, no more cases had developed; but on January 18, one of the birds had numerous proteosomata. It was also ascertained that many of these birds which previously had been found to be infected had now recovered, whilst others showed but a few proteosomata.

Thus twelve out of twenty-two birds (54 per cent.) became infected. This compares unfavourably with Ross's earlier results, as, in his published series, twenty-two out of twenty-eight (79 per cent.) were infected. But it is to be remembered that at the time this result was obtained the germinal threads were found at the end of a week; whilst in December the development was much slower, and took at least twice the time. It is much easier to keep mosquitoes alive during the first week after feeding them than it is to keep them alive for any subsequent period; moreover, in hot weather, such as Ross had worked in, mosquitoes bite more readily.

These results appear less unfavourable if they are considered in connection with observations on the normal proportion of wild, uncaged birds, infected with proteosoma at this season. Thus, earlier in the year, Ross, out of 111 wild birds, found proteosoma in fifteen, or 13.5 per cent.; whilst I found at this season only one out of thirty, or 3.3 per cent. affected with proteosoma.

It is possible that in the cold season the birds have a greater power of resistance; the validity of this conjecture is rendered more probable by the short duration of the proteosomal attack in my infected birds. Of the twelve, five died within the first week. In three of the survivors, in which the proteosomata had been very numerous, no parasites could be found ten days after the commencement of the invasion; in one in which they were never numerous none could be found on the fifth day. In the other three very few are now found, though at first they were numerous.

The recovery of these birds and the death of the mosquitoes fed on them diminishes the chances of much future work on this line during the time remaining to me here.

11. Mention has been made of the differentiation of the coccidia (previous to the formation of the germinal threads), according to the appearance of their contents, into clear and granular; the evolution of the latter into the coccidia containing germinal threads can be traced day by day. This differentiation was clearly visible in my series.

In a minority of the coccidia, and in most infected mosquitoes, when the germinal threads are mature, certain black tubular bodies are to be found in cysts with otherwise clear contents. These black tubular bodies were frequently met with in the series of mosquitoes infected in November and December. Most of these mosquitoes contained some coccidia

with black tubular spore-like bodies; though in a few insects all the cysts contained germinal threads only. In some cysts the black spores were numerous, and occupied the entire cyst; in other cysts there were only a few. In most instances germinal threads were not found in the black spore-bearing cysts, but there were a few such cysts in which it was doubtful whether germinal threads were present or not, or whether the appearance arose from over-lying threads which had escaped from a neighbouring capsule.

These black spores are very resistant. I have seen some which had been kept in water for months by Ross, and which had undergone no visible change. They withstand irrigation with liquor potassæ.

When the cysts are ruptured the black spores are to be found all over the body of the mosquito, but not included in cells. They do not seem to accumulate in any particular organ.

The most plausible view of the nature of these black spores seems to be that held by Major Ross, viz., that they are "resting spores," and that through them, by another cycle, the proteosoma can be propagated in conditions unfavourable for direct propagation by mosquito-insertion into a warm-blooded animal.

If this be the case, three courses suggest themselves:

(a) From the black spores may arise bodies capable of non-parasitic life (and possibly of reproduction), which at certain stages of their existence, and in certain conditions, on introduction into a warm-blooded host by inhalation, through drinking water, or even by injection by a mosquito or other blood-sucker in transferring them from the medium in which they live, may resume parasitic habits.

(b) That they may be ingested by mosquito larvæ, and in them undergo such development as will result in the formation of germinal threads in the adult mosquito, which, in turn, may be injected into the appropriate bird.

(c) That they may, if swallowed or inhaled by an appropriate warm-blooded host, so develop as to reach the circulation and pass into the sporulating phase.

Such experiments as have been made on this subject are inconclusive, and it is obvious that until the nature of these "black spores" is determined we cannot exclude, even for proteosoma of sparrows, the possibility of any one of the many possible alternative channels of infection. Intervention of the mosquito intermediate host may be only an occasional requirement.

Still less are we justified in concluding that malaria in man can only be acquired through and directly from the mosquito; or in devoting our attention exclusively to that channel.

(12) I have made myself familiar with the proteosoma in sparrows, and the halteridium in pigeons and crows.

In one specimen of a "blue jay," also, I found a very abundant halteridium infection; the parasites in this instance had some peculiarities which I hope to work out if we can procure more of these birds. The bird I had died before I had completed my observation; I have preserved the organs as well as specimens of the blood in the heart.

(13) In the cardiac blood of this jay there were numerous filariæ. They were sheathless, sharp-tailed and fairly active, and had locomotory movement. They were of two sizes; in the shorter the tapering of the tail was much more abrupt than in the longer. Neither showed any extension or contraction.

Adults of one species only, three females and five males, were found in the subcuticular connective tissue, and in that round the trachea.

They were much longer and thicker than *Filaria clava* (Wedl) or than the filaria described by Mazzini in the pigeon.

The females have the usual double ovary terminating in a vagina which appears tubular near the vulva situated near the caudal end of the body. The mouth is terminal and unarmed; the anus is sub-terminal.

The male has two spicules of equal length. The thickness of these worms, and the fact that when placed in weak formalin (2 per cent.) the cuticle burst in its entire length, will make them suitable for determining some of the disputed points in the anatomy of the Filaridæ.*

(14) The difficulties in connection with human malaria are increased by the present plague scare. The suspicion of the natives about inoculation makes them averse to any intercourse with European medical men.

By rewards, however, we have been able to get two fair cases of tertian fever, and three cases with crescent plasmodia—two of them with crescents in considerable numbers. On these cases we have fed mosquitoes—the common grey, and two varieties of "dapple wings" (large and small) in most points closely resembling those in which Ross had previously found pigmented cells after feeding on a patient with crescents. So far our results have been negative, but, in view of the peculiar climatic conditions, and of the possibility of the first stage, that of formation of coccidia, being inhibited by the cold, we are not prepared to accept these results as conclusive.

15. With Major Ross I have examined the organs of some persons (eight) who died of kala azar. This appears to be an infectious disease, indistinguishable at first from malaria. Chronic in character, it continues for months and becomes associated with enlargement of the spleen and liver, and progressive anæmia. The present opinion of most of those who have been deputed to investigate kala azar, as well as of those with longest and most intimate experience of the disease, is strongly in favour of the view that it is malarial in origin.

The melanin, or black pigment, was absent in the organs of some of the cases I examined; but in all but one yellow pigment was present in the liver, and in most in the kidneys and spleen also, indicating hæmolytic. The iron reaction with acidified potassium ferrocyanide was obtained in the spleen in three instances and, in one, in the liver also.

* Judging from the description of the embryos, it is probable that these blood-worms of the Indian blue jay are identical with those found by Manson in Amoy, China, in the magpie (*Pica media*) and the gray minna (*Gracupica nigricollis*), in which case the mature form of one will be found to lie in the pockets of the aortic and pulmonary semi-lunar valves (*vide Journ. of the Queckett Micro. Club*, vol. vi., p. 130, No. 44, August, 1880).

So abundant and chronic a hæmolytic in cases of malaria, continued moreover after the parasite has ceased to be present (at any rate in sufficient numbers to be found in the peripheral blood, or to cause appreciable deposit of melanin in the organs), raises the important question as to the possibility of the differentiation of parasites, with imperceptible morphological differences, by their toxic or hæmolytic properties.

16. Hæmoglobinuric fever seems to have been fairly common of late in some parts of India. I am collecting information, and have requested the editor of the *Indian Medical Gazette* to insert in that Journal a series of questions on the subject. Hæmoglobinuria does not occur in kala azar notwithstanding the great amount of hæmolytic which takes place in that disease.

I regret the length of this report, but the main subject of it, Major Ross' researches, cannot be dealt with in a few words, as they supply a basis for our future operations.

[It is necessary to point out that the word "coccidium" has been used by Major Ross and in Dr. Daniel's report above printed in a peculiar and not readily intelligible sense. "Coccidium" is the name of a genus of Sporozoa, established by Leuckart in 1879 for the cell-parasite of the rabbit's liver, called *coccidium oviforme*, and other allied species. "Proteosoma" is the name given by Labbé to another genus of Sporozoa parasitic in the blood-cells of birds. When Major Ross states in his report, dated May 21, 1898, that certain "parasites are a development in the mosquito of proteosoma in birds; and to judge from their structure and mode of growth so far as yet observed, I take them to be coccidia," he is using the generic term "coccidium" to describe some phase in the growth of the species of a distinct genus, *Proteosoma*.

Apparently, what Major Ross intends to indicate by the term "coccidium" is an ovoid firmly walled corpuscle which increases in volume from about $\frac{1}{1000}$ inch in length to four or five times that size, and then breaks up into a mass of filiform spores radiating from a central granular mass.

In this mode of spore formation these bodies have resemblances to the true coccidia, which present themselves not only as oviform corpuscles but as cysts with sickle-shaped or filamentous spores. It is, however, not legitimate to apply the generic term "Coccidium" to a phase of growth of another genus. —*LISTER, Chairman of the Malaria Committee.*]

ANKYLOSTOMIASIS IN BELGIUM.

According to the French Consul at Liège a severe epidemic of the above disease is raging among the working population of that neighbourhood, especially miners. Since its discovery by Dubini at Milan in 1888 *Ankylostoma duodenale* has been found in nearly every country of the globe between 30° south and 51° north latitude, though it decidedly prefers the tropics. Griesinger showed it to be the cause of Egyptian chlorosis, but it first attracted European attention in 1890 when it gave rise to an epidemic of anæmia among the workmen employed on the St. Gothard tunnel. Similar epidemics have occurred in various parts of Europe, usually among miners and brickmakers. (*Revue Scientifique*, No. 22.)—*Janus*, July and August.

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

CASES OF OCULAR LEPROSY.—Dr. J. Bistis, of Constantinople, publishes notes of five cases of ocular leprosy in the May number of the *Archives d'Ophthalmologie*. Of these, two are cases of leprosy iritis, one of paralytic lagophthalmos, one of stricture of the lachrymal duct owing to extension of leprosy infiltration from the nose, and one of a corneal leproma, so large as to prevent the closure of the lids. The writer has seen nineteen cases of ocular leprosy in all; in twelve of these he was able to explore the fundus, but could detect no changes in the retina or choroid; he expresses scepticism as to the leprosy nature of the fundus lesions described by Trantas, and draws attention to the fact that Bull and Hansen found the fundi normal in two hundred lepers. In all his cases the ocular lesions (excluding lids) were among the later manifestations of leprosy, appearing between the eighth and fifteenth years after infection. Tubercular leprosy is the form which most frequently affects the eye. Lesions of the cornea and conjunctiva seen in anæsthetic leprosy are generally due to exposure from lagophthalmos, and only indirectly to leprosy. He advocates the removal of corneal lepromas when large; the growth of small ones may be arrested by marginal keratotomy. Cataract extraction is not contra-indicated in leprosy.

TRACHOMA AND RACE.—My article in the *British Medical Journal* of May 6, drawing attention to the immunity and partial immunity of certain races as regards trachoma—an immunity in no way dependent on climate or sanitary surroundings—has elicited an interesting letter from Dr. Freeland, of Antigua, on the subject (*British Medical Journal*, July 1). Dr. Freeland has found trachoma very common among West Indian negroes, in marked contrast to the state of affairs in the United States, where true trachoma is almost unknown amongst the coloured population. I have written to Dr. Freeland asking for further information on certain points, and postpone comment until I receive his reply.

Within the last few days I have had the opportunity of conversing with Dr. E. Oliver Belt, surgeon to two large hospitals in Washington, the patients in which are mainly negroes (Freedmen's Hospital, and Episcopal Eye, Ear and Throat Hospital). His experience bears out that of other American observers, viz., that trachoma is very rare amongst the coloured population, and practically unknown in pure-blooded negroes. Cases are so rare, in fact, that they are shown at the ophthalmological societies as curiosities.

Dr. Van Millingen's opinions on this subject, and the vigour and ability with which he defends them, are well known to ophthalmic surgeons, and I am glad that my article has been the means of eliciting an expression of them for the benefit of the readers of the *JOURNAL*. As regards the controversial portion of his valuable paper, I must postpone replying to his strictures on the views I tentatively expressed, as I hope to be in possession of fresh facts and statistics, and to be in a position to reply on the whole subject. Meanwhile we must all welcome with unalloyed pleasure Dr. Van Millingen's first contribution to the *JOURNAL*, which it is to be hoped is only the precursor of others: his reputation as an ophthalmic surgeon is an international one, and his long residence in the East enables him to speak with authority on the many interesting problems connected with tropical ophthalmology.

I have received a private letter from M. Chibret, of Clermont Ferrand, the pioneer in the work of elucidating the connection between trachoma and race, and cannot forbear from quoting one or two of his remarks:—

"It is, I believe, absolutely certain that the factor which dominates the etiology of trachoma is the factor of race You are then to be congratulated on having brought this fact into prominence in England, as it may

serve to elucidate many problems, not only of medicine, but of anthropology. Receptivity to trachoma may come to be looked on as a racial characteristic, and serve to differentiate races in other respects alike."

M. T. YARR.

GERMANY.

METHYLENE BLUE IN ACUTE ARTICULAR RHEUMATISM.

Lemoine has found from considerable experience that methylene blue is a very effective remedy in acute articular rheumatism. He believes it is more rapid and efficient than sodium salicylate, especially in that of gonorrhoeic origin.—*Bull. de l'Acad. de Méd.*, January 31.

AMERICA.

THE BACILLUS OF INFLUENZA.

Drs. Reilly and Wyncoop have been investigating the causal relation of the influenza bacillus to the disease, and details of their work appear in the *Bulletin of the Chicago Bureau of Vital Statistics* for January, 1899 (summarised in the *Medical Age* for March 25). The questions they set themselves to solve were: (1) Is there a true influenza caused by a special germ, and (2) is there a pseudo-influenza also, in which this specific organism is absent? Dr. Wyncoop's investigations have shown him that Pfeiffer's bacillus is easily identified in the patient's sputum, and it is therefore not necessary to go through the process of cultivating it in the laboratory. The result of his work goes to prove that true influenza is widely prevalent. Many cases which at first appeared to be cases of diphtheria were due to infection from Pfeiffer's bacillus, and the subsequent clinical developments confirmed the microscopic diagnosis of influenza. Many cases which appeared to be examples of simple tonsillitis or laryngitis showed the presence of Pfeiffer's bacilli in the expectoration. He also states that many physicians have observed that when influenza was present in a house the eyes of those members of the family who were not affected by the disease became suddenly and acutely inflamed, and were the seat of copious purulent discharge. Cultures made from these discharges showed the presence of large numbers of influenza organisms.

Though the diagnosis of the disease can easily be made by microscopical examination alone, yet cultures can be made to verify the diagnosis in from ten to fifteen hours. By these two methods of investigation an absolutely reliable diagnosis can be obtained.—*Scottish Medical and Surgical Journal*, July.

Communications, Letters, &c., have been received from:—

- A.—Dr. Izett Anderson (Eastbourne).
- B.—Dr. Osborne Browne (British Honduras).
- C.—Dr. H. Chassaud (Smyrna); Dr. P. T. Carpenter (British Honduras).
- E.—Dr. P. G. Edgar (Perah).
- L.—Mr. John de Leon (Jamaica).
- M.—Mr. C. G. Moor, M.A., F.I.C. (Exeter); Dr. McDowell (Nigeria); Mr. C. H. Massiah (Demerara).
- P.—Fleet-Surgeon E. R. H. Pollard (Queenstown); Dr. Wordsworth Poole (Westminster); Dr. W. G. Tottenham Posnett (Dublin).
- R.—Dr. Palmer Ross (London).
- S.—Dr. H. C. Sherman (Korea).

EXCHANGES.

Annali di Medicina Navale.
Archiv für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.

Bristol Medico-Chirurgical Journal.

British and Colonial Druggist.

British Journal of Dermatology.

British Medical Journal.

Clinical Journal.

Giornale Medico del R. Exercito.

Il Policlinico.

Indian Engineering.

Indian Medical Gazette.

Indian Medical Record.

Janus.

Journal of Balneology and Climatology.

Journal of Laryngology and Otology.

La Grèce Médicale.

Lancet.

Liverpool Medico-Chirurgical Journal.

Medical Brief.

Medical Missionary Journal.

Merck's Archives.

New York Medical Journal.

Pacific Medical Journal.

Polyclinic.

Public Health.

Revista Medica de S. Paulo.

South African Medical Journal.

The Hospital.

The Medical and Surgical Review of Reviews.

The Northumberland and Durham Medical Journal.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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THE KNOWN AND THE UNKNOWN IN RESPECT OF BERIBERI.

By MAX F. SIMON, M.D.

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It has struck me that the JOURNAL OF TROPICAL MEDICINE, which, it is to be presumed, has a large circulation among practitioners in the tropics, might be advantageously used as a record of combined research in points connected with tropical disease as to which definite knowledge is still lacking, were such points clearly brought to notice in its pages. With this end in view, it would be well if medical men would contribute papers on certain diseases, *e.g.*, dysentery, abscess of the liver, sprue, or others in which they have experience, setting forth clearly what is definitely known about such diseases, and what is as yet unknown, and therefore desirable to be ascertained. It must be a matter of common observation how often the same thing is noticed by different practitioners, and published over and over again in papers which, in truth, have every claim to originality, while points on which exact information is urgently required are allowed to remain *in statu quo*. I propose, in the following remarks, to endeavour to state shortly the points in relation to the disease known as beriberi which may be considered settled, and therefore may be called "known," and the points on which definite knowledge is still required, and which may therefore be called "unknown."

ETYMOLOGY.

No further knowledge is required as to the etymology of the name of the disease. After a long period

of ingenious endeavour to find its derivation in words of Sanscrit or other languages, the simple fact is now recognised that "beriberi" is a Sinhalese word and means "a bad sickness." The pathology and symptoms have been fully described; it is well known, to put it shortly, that beriberi is a peripheral neuritis, that its symptoms depend upon the particular peripheral nerves attacked, and that its danger to life lies in the proclivity of the peripheral nerves to certain important organs to attack. The *post-mortem* appearances, macro- and microscopic, have been fully described.

WET AND DRY VARIETIES.

It is known that the old division of the disease into two varieties, the wet and the dry, is not tenable, but that the two conditions may exist together, either may exist alone, or either may supervene on the other at any time during the course of the disease. It is known that, so far as causation goes, beriberi has nothing to do with *anæmia*, or *ankylostomiasis*; that, so far as any association with these conditions may be found to exist, such association is accidentally coincident. The general principles of treatment of the disease are also I think well known; they consist essentially in a treatment of symptoms.

THE INFORMATION WANTED.

The points on which definite knowledge is chiefly wanted are points in connection with (a) prophylaxis, (b) the cause, or causes of the disease, and (c) the question of its communicability from person to person.

PROPHYLAXIS.

(a) As regards prophylaxis, I am inclined to think there is a good opening for investigation as to whether a great deal cannot be done. Hitherto attempts in this direction have been chiefly dietetic or hygienic,

and have consisted in improvement in diet and change of air. In certain cases, as in the Japanese navy, improvement in diet has been followed by a marked decrease in the disease, but there is no doubt that improvement in diet, in this case, was accompanied by improvement in hygienic matters, and it is difficult to say how much good was due to one thing and how much to the other. In other cases, improvement in diet, where much improvement in hygienic conditions was not needed, has not been followed by marked success.

THE USE OF FAT.

I have lately seen in the *Lancet* a most encouraging statement as to the influence of the administration of fat with food in controlling and stopping two outbreaks of beriberi, but its occurrence among Chinese prisoners in whose dietary fat pork has a somewhat prominent place seems to indicate that this line of treatment would not be always successful. Change of air has really perhaps been found more useful in the treatment of those already ill than as a prophylactic, but in many cases in which one might like to try it in the latter capacity, circumstances may not permit of such trial.

DRUGS AS PROPHYLACTIC AGENTS.

It is to the possibility of there being any drug which may be used prophylactically against beriberi that I think attention may be profitably directed, and this line of research might be usefully taken up by medical officers who have charge of bodies of men, in gaols, at mining work, or under other conditions, among whom the disease occurs. Until treatment by antitoxination of any disease is fairly reliable, the best possible must be done without it, and this best will probably be found in the pharmacopœia. It is not necessary for the trial of any particular line of treatment to be followed by success that should prevent the fact of such trial having been made from being published; such publication would probably prevent unsuccessful work being done several times over.

ARSENIC.

I had for a long time an idea that arsenic would prove useful in this direction, and have lately had an opportunity of trying it on a large body of men among whom cases of beriberi have been constantly occurring for a long time—the result was quite unsuccessful.

STRYCHNINE.

Of late, Dr. Ellis, medical superintendent of the Lunatic Asylum in Singapore, reasoning that, as strychnine is of very great value in combating some of the fatal conditions of the disease, it might therefore act also in lessening the liability of such conditions to be caused by the poison, has been trying this drug with prophylactic intent. He gave to every patient in the Asylum, with the exception of a few who would take no medicine, first three, then five, and afterwards ten minims of liquor strychninæ thrice daily. At first results seemed to be very promising, especially in respect of reducing the death-rate, but the early promise has not been fulfilled lately and the trial has been given up.

ÆTIOLOGY.

(b) In regard of the second of the points I have mentioned, the cause, or causes of the disease, definite knowledge is much required. In addition to defective hygiene and insanitary environment in endemic areas, the immediate cause of beriberi has been credited to bacteria (Pekelharing's diplococcus), to deficiency in nitrogenous food, to malaria, to parasitic disease of rice, to particular kinds of rice, and perhaps to other things; but none of these causes can be made to fit in with every outbreak. It is now generally received that the immediate cause of beriberi is a toxin (possibly bacterial in origin), and all evidence points to the fact that this toxin is manufactured outside the body. A careful analysis therefore of the conditions under which beriberi occurs is required, and a gradual determination, by a process of elimination, of those that are absolutely necessary.

ENVIRONMENT FAVOURING OUTBREAKS.

The conditions of environment that are generally described as favourable to outbreaks of beriberi, or as being present where beriberi is endemic, and favouring the manufacture of the toxin, are roughly as follows:—Low-lying, damp, or marshy situations, accompanied by overcrowding, defective ventilation, general absence of sanitation, and bad food; and it is quite true that beriberi occurs in places in which all these conditions are found. It is equally true, on the other hand, that it occurs in places which are neither low-lying nor damp, that are well-ventilated and sanitary (so far as they can be made so), where there is no real overcrowding, and where every attention has been given to the quantity and quality of the food. As examples of what I have stated, I may name as places where the disease is endemic, or where outbreaks have occurred, or are occurring, and where in some cases all, in some cases a few, in other cases apparently none, of the conditions above mentioned are found—certain districts in Yokohama; mining and other districts in the Malay peninsula; on board of ships; among patients admitted for other diseases in Tan Toeh Sing's Hospital, Singapore; and in the Pauper Hospitals at Penang and Malacca; in the Lunatic Asylum, Singapore; in the gaols in some of the Native Malay States; in the old gaol, Singapore; in the present gaol, Singapore; and in the General Hospital, Singapore.

ONE CONDITION CONSTANT.

In all these places, so far as I can make out, there is only one condition which is *absolutely constant* in all, viz., that persons are together in considerable numbers, not even over-crowded. It is worthy of note, in this connection, that, so far as I know, beriberi does not occur, and has never occurred, among the thousands of Indian immigrant coolies who inhabit the extensive coolie-lines on the estates in Province Wellesley; yet Indians are not exempt from the disease, nor is Province Wellesley exempt from the disease, for it has broken out among Chinese coolies living also on sugar estates in the district.

THE TOXIN MAY VARY.

I have myself been constantly familiar with beriberi since about 1879, that is, for twenty years, and, after

having been a convert, in turn, to most of the various views that have been advanced as to its etiology, I now think the disease to be as great a puzzle as ever. I do not even feel sure, taking into consideration the analogy of neuritis from alcohol, arsenic, lead, malarial intoxication, leprosy, and perhaps other causes, that the toxin that produces beriberi is always the same, or that, in dealing with different outbreaks of beriberi, we are dealing always (though symptoms may be identical) with the effects of the same poison, but feel inclined to doubt, sometimes, that there is any such one real specific disease-entity as beriberi at all. With regard to malarial neuritis, the writer of an editorial in a comparatively recent number of the JOURNAL OF TROPICAL MEDICINE takes exception to the term as implying that malaria can cause neuritis, which, he says, it cannot do, any more than it can cause scorbutus, but that it can only co-exist with it as one part of a dual infection. It is possible, however, that "malaria" may comprehend more than the parasite or parasites which are now recognised as the cause of certain periodic fevers.

COMMUNICABILITY.

(c) The question of the communicability of the disease from one individual to another is one that requires to be definitely settled. There is no doubt, though in most outbreaks evidence points to the disease being contracted from an infected place, that many instances occur in which the possibility of contagion, as generally understood, may very well be entertained. The first thing to be decided here is the *possibility* of communicating the disease, and for this it is necessary to know where the poison is to be found; and here this question is mixed up with that of the cause of the disease. Attempts at bacteriological cultivation have been made by the hundred, with, so far as I know, little, if any, result beyond the growth of a coccus of very doubtful powers. The only experiments that have been done as to inoculation with this coccus are those of Pekelharing and Winkler, which were not conclusive. From the results of an experiment which I made many years ago, but which I have never had opportunity to repeat, I am inclined to think that neuritic results will be produced by inoculation with the serum of a beriberi patient. If this be found to be correct, the next step will be to try inoculation with the filtered serum; then, if neuritic results follow, bacteria as such will be put out of court, the toxin theory will be established, and also, I take it, the non-contagiousness, in the ordinary sense, of the disease. There seems to be a good amount of evidence to show that beriberi may be introduced into communities, in gaols, asylums, and the like, by the admission thereto of patients suffering from the disease, so that it is probable that the poison, or means for its manufacture, can be given off in some way from the body. The late Dr. Hampshire, formerly Colonial Surgeon at Penang, was always of opinion that beriberi is propagated through the excreta of patients somewhat after the manner of enteric fever.

I have endeavoured, in the above remarks, to indicate some points to which research may be advantageously directed. Except in the direction of cul-

tivation experiments, I do not know that the blood in beriberi has been much studied, or that anything more definite has been chronicled than that there is no lessening of number of red corpuscles, no change in number of white, but generally a deficiency in hæmoglobin to the amount of from 15 to 20 per cent;¹ and it seems to me possible that considerable help in the solution of the problem of the cause of the disease may be obtained from pathological chemistry.

BERIBERI IN STRAITS SETTLEMENTS.

The importance of the disease here may be judged by the following figures, which give the numbers of cases of beriberi admitted, and of deaths from it, in the Government Hospitals of the Straits Settlements during the last five years:—

			Admissions		Deaths
1894	1203	...	161
1895	1594	...	435
1896	2057	...	730
1897	2058	...	685
1898	1329	...	420

These figures also show that these Settlements, with the Federated Malay States, where the disease also occurs to a considerable extent, would afford an unrivalled field for study, in case any holder of a Research Scholarship, or Travelling Fellowship, should feel inclined to investigate a disease which Dr. Manson, in his recent work on "Tropical Diseases," wisely classifies as a general disease of undetermined nature.

TUBERCULOSIS IN INDIA.

By MAJOR W. J. BUCHANAN, I.M.S., B.A., M.B.
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THE great amount of attention which is now in Europe being given to the prevention of tuberculosis makes the question of its prevalence in India and the tropics generally one of more than ordinary interest. On the threshold of any such inquiry we are met with the doctrine of an antagonism between phthisis and malaria. It is therefore necessary to briefly examine this point and to see how far it is a fact.

RECORDED ANTAGONISM BETWEEN MALARIA AND PHTHISIS.

The doctrine of an antagonism between malaria and phthisis is an old one. In England, in 1811, a Dr. Wells was the first to make the pronouncement; and in 1846, the celebrated French writer, Boudin, definitely formulated his views that pulmonary phthisis was very rare in marshy countries, and that in localities where phthisis was common malaria was slight, or, in other words, that the cause of malaria (we would now say the parasite) produced such a modification of the human organism that it became refractory to tuberculosis. Many other French writers have noted that phthisis is rarer in Algeria than in France. Collin, however, could find no antagonism between paludism

¹ Ellis, *Lancet*, October 15, 1898.

and phthisis. He had often found, he wrote, tuberculosis in persons saturated with malaria. Laveran's experience is worth quoting; out of 69 fatal cases of disease in his hospital in Algeria, during three years, no less than 9 were due to tuberculous affections, a proportion which, however, he says is inferior to that of the French army at home. He also (*Traité du Paludisme*, p. 246) gives figures to show that the foreign troops (Chasseurs d'Afrique and Zouaves) suffer from phthisis less than half as much as a pure French regiment. This comparative rarity of phthisis does not, however, hold good in all malarial countries. Laveran quotes the statement that at Walcheren one-fourth of the deaths are due to phthisis.

RECORDS SHOWING PRESENCE OF BOTH MALARIA AND PHTHISIS.

In Greece, Boussakin found tuberculosis very common in the most malarious villages. In Tonquin both malaria and phthisis are very common. So also in Guiana and in the Antilles. On the other hand, Virchow hardly ever found a case of phthisis in Silesia, where nearly every one had enlarged spleen. Again, Dr. R. W. Felkin, on his journey from Khartoum to Lake Albert, found agues very common and phthisis very rare. The latter, however, was found in Shuli and other places at an elevation of 3,000 feet. The fen country of Lincolnshire was said to have very little phthisis, while agues used to be prevalent (Haviland). In Siam both diseases are common; in Corea ague is common and phthisis said to be unknown. We have Hirsch's authority that consumption prevails to a disastrous extent among the races of the South Pacific, especially in Fiji, Tonga, Hawaii and among the Kanakas, and it is by no means certain that it was introduced by European immigrants (Hirsch, vol. iii., p. 187). Phthisis is said to be common among the Hottentots. Negroes in their own land are said to escape, but tubercular diseases are very common among them when removed to other countries, as the United States, West Indies, Brazil or Ceylon. In American prisons from 1829 to 1841, the death-rate of negroes was 40 *per mille*, compared with 11 *per mille* among whites. This, we must believe, was in part due to the very inferior sanitary surroundings provided in those days for the negro prisoners. In the West Indies phthisis has been noted as common among East Indians who have migrated thither.¹

From the above often contradictory statements it will be seen that, although the known facts do not amount to any antagonism between phthisis and malaria, yet it is the fact that in nearly all warm countries phthisis is less prevalent than in cold northern climes, but this partial immunity is due to general causes (open-air life, sleeping in verandahs, use of mat and reed huts, &c.), rather than to any individual resistance. The worst and most rapid forms of consumption may be seen in malarial cachectics. The terrible ravages of phthisis among tropical peoples when removed to other or colder climes is no proof of a special susceptibility to the disease, it only illustrates the law laid down by Dr. G. Archdall Reid, "that the resisting power of

any race is exactly proportionate to its familiarity with the disease."¹

We must now refer to our special subject, the prevalence of phthisis in India. For the vast majority of the population we have no statistics for tuberculous disease. We must therefore rely upon the carefully recorded statistics of three classes of the community, viz.:—European troops, Native troops, and Native prisoners, amounting on the average to a daily strength of 300,000 in all.

Two questions are here to be considered: first, the incidence of tuberculosis upon Europeans after arrival in India; and secondly, among the natives of India.

TUBERCULOSIS AMONGST EUROPEANS IN INDIA.

In the first place, as regards European troops it is satisfactory to note a marked decline in the prevalence of phthisis. The admission to hospital rate for British troops in 1897 was only 4·2 *per mille*, whereas in the period 1871 to 1876 it was more than double, or 9 *per mille*. In all India the death rate *per mille* of strength of British troops in 1897 was only 5 *per mille*, but it varied from 1·12 in Burma to a very small decimal in Central India and the Deccan. It was in fact lower in India than the average for the troops at home (7 *per mille*). Much of this must be attributed to improvements in the ventilation and sanitation of barracks.²

As regards the prospects of a young European landing in India with incipient consumption, we may say that India, if not a bad place for a healthy man, is a very poor place to convalesce in. The extremes of climate, heat and cold, moisture and dryness, are too great for any but the healthy. During the delightful dry cold weather progress would be made, but it would be lost, if not in the fiery hot season, certainly in the hot rainy season. It appears, on the whole, that the damper parts of India are worse for phthisical European patients, *e.g.*, Burma and Bengal. I have known two cases of undoubted phthisis in young Europeans completely recover in India, but they both lived in a dry, if hot, climate. Another point to be considered in favour of Europeans *predisposed* to tuberculosis is that the majority of them live a healthier, more open-air, out-of-door life than would be possible in ordinary town or city life in England. The houses are larger, with infinite facilities for ventilation, doors or windows are open day and night for the great part of the year. Norman Chevers noted this fact and wrote, "I believe the immunity from phthisis of well-to-do Europeans in Bengal is mainly due to the free ventilation of their sleeping rooms." As regards the value of hill-stations for cases of tubercle of the lungs, the opinions formulated many years ago by Dr. (now Sir Joseph) Ewart are still in accordance with experience. Hill climates suit scrofula. They are useful as an escape from the heat to mild slow cases of consumption, or to cases on the mend, but they are injurious at all seasons, and

¹ "The Present Evolution of Man," p. 292.

² Invaliding much reduces the death rate for all chronic diseases among British troops in India. In 1897, of 282 cases of phthisis, 37 died, but 133 were invalided home as unfit for further service in the army.

¹ Grieve, *Brit. Guiana Med. Ann.*, 1890.

especially in the rainy season, to all active or advancing cases of consumption.¹

In my experience cases of tubercle of the lungs in Europeans unless arrested run a very rapid course in Bengal. If it was not for the heavy rains from June to September, the numerous hill stations in India would form admirable places for trying the open-air treatment.

TUBERCULOSIS AMONGST NATIVES OF INDIA.

We now come to a consideration of the prevalence of tuberculous affections in the natives of India. We have already referred to the supposed antagonism between tuberculosis and malaria. My own experience leads me to believe that tubercle of the lungs is more common in natives than is generally supposed, and that many cases are overlooked in out-patient practice. The patient generally complains only of "fever;" emaciation has not the same significance in an Indian hospital as it would have in England. Cough² is much less commonly a prominent symptom of phthisis in India. I pointed this out six years ago in an article³ on Tuberculosis in Bengal Jails. I have recently come across the following remarks on phthisis in India in an old article in the long extinct *Indian Annals of Medicine* (No. xxi.) by Dr. (afterwards Surgeon-General Sir Wm.) Hoore, who wrote:—"Phthisis, formerly supposed to be so rare among natives of India, is now admitted to be a very frequent affection, and particularly so among inhabitants of the Upper Provinces (see Wilson, also Webb's *Pathologica Indica*). The disease is frequently somewhat different to what has been observed in England. The extent to which tuberculous disease occurs in coloured races has become evident from *post-mortem* rather than direct diagnosis."

TWO FORMS OF PHTHISIS IN INDIA.

"There appear to be two forms of phthisis in India, the latent and the febrile. The former runs its course without any symptoms excepting emaciation, without cough, expectoration, or hæmoptysis, suddenly then febrile symptoms set in. Emaciation should always excite suspicion and lead to stethoscopic examination. In over-crowded jails phthisis is excited; it not infrequently assumes a more or less latent form, and when febrile symptoms present themselves they may be mistaken for malarious." Dr. Hoore also remarked that he had often met delicate Europeans with undoubted tubercular deposit in the lungs who suffered neither from cough nor expectoration, but simply from evening hectic and more or less emaciation. Chevers' experience is to the same effect. "Cough," he wrote, "does not of necessity attend phthisis."⁴

This is exactly my own experience, arrived at independently seven years ago. In prisoners, loss of

weight often calls attention to commencing tubercle in persons without any special symptoms directed to the lungs. Again, it is not uncommon to see a patient with intense anæmia, enlarged spleen, pearly blue-white conjunctivæ, and irregular fever, such as one might easily mistake for malarial cachexia. If such a person's lungs be examined evidence of often advanced tuberculosis may be found, though there has been no regular hectic, nor any cough to attract attention. I confess to having made the diagnosis of more than one such case only on the *post-mortem* table. In an article read before the British Medical Association at Plymouth, I gave several cases of what I call "terminal dysentery" occurring in tuberculous cases, that is dysentery supervening when the patient was *in extremis*, a few days before death. Several of these cases showed tubercle of the lungs in association with malarial cachexia. In 1898 I published a case in which general tuberculosis¹ simulated malarial cachexia, so that the case was "returned" under the latter heading, till I happened to examine the lungs, when the state of affairs became clear and the diagnosis of tubercle was amply confirmed at the autopsy. The following case (No. xxii.) from my list of "terminal dysentery" cases may be here quoted:—"Chowkidari Dome died February, 1898, from tubercle of the lungs. Much emaciation, both lungs broken down. The pericardium contained 2 oz. fluid. Much ascites, spleen much enlarged. Small intestine healthy, large intestine showed recent low gangrenous inflammation. The symptoms of bowel complaint only came on within past twelve days. An example of tubercle occurring in a malarial cachectic, and of 'terminal dysentery.'"

I have said above that I consider tuberculosis a not uncommon disease in India. It is possible perhaps to reduce this statement to figures. Out of 10,650 deaths from all causes among 236,000 native soldiers and native prisoners in the years 1896-7, we find no less than 863 deaths from tuberculosis (all forms), that is 8 per cent., or 1 death from tubercle in every 12. The ratio in England is said to be 1 in 7, so that the difference is not great when we consider that the Indian figures do not apply to all ages of the population. One death in twelve is a pretty high percentage for one disease only of all the ills that flesh in the tropics is heir to.² How far it will be possible to reduce this ratio in the future remains to be seen. Norman Chevers, years ago, proclaimed

¹ *Indian Medical Gazette*, 1898, p. 139.

² The following lists give approximately the order of fatality of the chief diseases in India:—

European Troops	Native Troops	Native Prisoners
Enteric fever	Pneumonia	Dysentery
Abscess of liver	Malarial fevers	Pneumonia
Cholera	Tuberculosis	Cholera
Dysentery	Cholera	Diarrhoea
Malarial fevers	Dysentery	Tuberculosis
Heat apoplexy		Malaria
Tuberculosis		
Pneumonia		
Heart diseases		

¹ *Trans. of Epidem. Soc.*, vol. i., p. 26.

² In the *Practitioner*, July, 1899, p. 17, Dr. Philip, of Edinburgh, writes:—"Cough need hardly be present. It is the prayer or the groan for more air; if an unlimited supply of fresh air is given it need not be heard." This, perhaps, partly explains its absence in many Indian phthisis cases. — W. J. B.

³ *Indian Medical Gazette* for 1893 (August).

⁴ "Diseases of India," p. 526. See also quotation (*loc. cit.*) from Goodeve, about the commonness of tuberculosis in Indian hospitals.

the value of fresh air and good ventilation at a time when men in England were deaf to the voice of Mac-Cormac, and were still wrapping up their phthisical patients lest the winds of heaven should visit their faces too roughly. At the present time I have a patient, a prisoner, a Bengali of good family, who was sent here in an advanced stage of phthisis for a change. He then weighed only 107 lbs., and had irregular hectic fever, night sweats, and had had several attacks of hæmoptysis. I put him on light labour, out of doors, clothed and fed him well, made him walk about or sit out of the house for several hours daily, made him sleep in a roomy ward with ten open doors or windows, with the result that after one year he has gained 21 lbs. in weight, looks fat and sleek, has had no fever, sweats, or hæmoptysis for over six months; in fact, no one now examining him would suspect phthisis. If this can be done within a prison's walls it can certainly be done outside. The great difficulty, in my opinion, would be in India to educate even the better-class native to appreciate the value and necessity of free ventilation and open doors and windows.

TUBERCULAR AFFECTIONS OF BONES, JOINTS, &c.

We must now say a word about other tuberculous diseases. Among European soldiers we find that only 14 per cent. of all tuberculous diseases are affections of the glands, bones and joints, and this falls to 9 per cent. among prisoners, and only 5 per cent. among native soldiers. More remarkable still, out of 236,000 native troops and convicts in 1897 there was not a single admission for tuberculous disease of either bones or joints. Some cases, of course, would be included under the heading Tubercle of the Lungs, but still, the fact remains that there is comparatively little bone or joint tuberculous disease in India, that is, contrasted with European experience. Native women of the higher class, immured for the greater part of their lives within the walls of the zenana compound, are said to suffer considerably, however, from tuberculous affections of joints and bones; but men certainly suffer comparatively little.

We may conclude that even if tuberculosis figures to a slight degree among the list of admissions to hospitals in India, still it looms largely in the death returns. A disease which carries away one patient in every twelve in a malarious country can scarcely be said to be antagonistic to malaria, or to have malaria antagonistic to it.

THE PUERPERIUM IN THE TROPICS.

By W. D. SUTHERLAND, M.B.

Capt. I.M.S., Civil Surgeon, Sangor C.P., India.

NOT all the *ex-cathedra* statements which we so gladly received and unhesitatingly believed in our student days have stood the searching tests of time and experience; and among those, which having been weighed in the balance and found wanting are now relegated to the limbo of things best forgotten, fall to be counted three obstetric *obiter dicta*.

How many of us have treasured the very sound of

"perfect rest," "tight binder," "antiseptic douche." The idea used to be that in order that it might be well with us and our parturient patient, before all things it was necessary that we should (1) apply a binder to the patient's abdomen drawing it as tight as possible, (2) keep her absolutely at rest during the puerperium, and (3) douche the genital canal regularly and frequently.

Then if she had no rise of temperature we might congratulate ourselves on having done our duty; if she did have a rise of temperature the douche must be used more energetically.

Now this is not amusing, far from it. At the very best of times the puerperium is not a pleasant episode in the life of a woman in the tropics, and shall we make it more pleasant by keeping her, swathed like a mummy, lying on her back, and periodically flooding her with pints of a solution of this or that fashionable "antiseptic?" I trow not; yet this is practically what used to be the regimen, without which there was no salvation.

THE BINDER—IS IT BENEFICIAL?

Were all this as unavoidable as it was said to be there would be nothing for it but to carry out the treatment to our own as well as our patient's discomfort. But is it unavoidable? I think not, as I have said. To begin with the binder, this modification of a cere-cloth is advocated on the following grounds: it is said to "preserve the figure"; to prevent any unpleasant results following the sudden evacuation of the abdominal contents; to promote contraction of the emptied uterus, and even to prevent relaxation of a uterus which is now contracted.

Such, more or less as I have set them down, are the reasons advanced by those who are in the habit of pinning their faith on the binder. Let us examine them. The argument regarding the preservation of the figure amounts to a statement that the application of a tightly constricting cloth to a woman's abdomen will prevent pendulous belly. Doubtless it will do so, for just as long as the binder is worn! But, in the name of common sense, how can a temporary constriction of the belly have such an effect when once the binder is taken off? By promoting a peculiar condition of the abdominal wall? As if constriction of the abdomen did not cause increased pressure upwards into the thorax. So much for *a priori* reasoning; now for the significant fact that even a fat multipara does not develop pendulous belly as a result of never having had her figure preserved.

Again, the binder is said to obviate the untoward consequences of the sudden emptying of the uterus which takes place at parturition, and causes a change in the intra-abdominal pressure. This is, of course, a relic of the idea that any thing which caused a change within the sacred penetralia of the abdomen was bound to be of bad effect. The old surgeons had harrowing tales to tell of the awful consequences of the sudden evacuation of an ascitic collection of fluid, and, doubting the fact that a poor wretch, whose heart and kidneys were such that his abdominal vessels had to throw off their load, *did* faint, greatly increased their superstitious respect for the peritonæum. But surely a healthy parturient woman is not quite like a patient

in the last stage of dropsy! When all is said and done, child-bearing is a natural act, and even if it did—as it does not—occur in the twinkling of an eye, it is likely that nature would have a way out of the difficulty. This *à priori*; and here again experience proves that the binder is not required.

But we have the solemn assertion that the uterus will contract better when a binder is applied; nay, more, some even place a large pad over the uterus under a binder to facilitate this contraction.

We know that friction of the uterine wall, through the abdominal parietes, will cause contraction of the organ as a whole; but how mere pressure, which, as I have hinted, does not exert its influence more on the pelvic viscera than it does upwards on the thoracic contents—how such pressure can be of service in promoting contraction of a hollow muscle like the uterus, or in keeping it from relaxing, when once it is contracted, I cannot for my life see. And experience teaches that the uterus contracts and remains contracted *without* the binder.

IS REST IN THE HORIZONTAL POSITION OF BENEFIT?

Now as to rest. This is, of course, a great desideratum for the patient; but surely some form of it may be permitted which does not entail her lying absolutely motionless on her back. This is a most irksome position, even for one seriously ill, and it favours stagnation, and consequently decomposition of the lochia.

That the dorsal decubitus does *not* favour the emptying of the vaginal canal we see every time that we give a vaginal injection. As soon as the woman turns round or gets up, there is a gush of fluid, after all had apparently drained away. As to this posture being irksome, we need only ask the patient whether she would not like to lie on her side, and hear her eager "Oh yes," to make up our minds on this point.

THE ANTISEPTIC DOUCHE.

The question of the antiseptic douche would seem to be one regarding which there can be only one opinion, and that distinctly in its favour. Doubtless it is of great value, when the patient is condemned to the supine decubitus, in promoting removal of the lochia, and thus preventing their decomposition. On the other hand, when the patient is allowed to lie as she likes, and made to turn round on to her face when she micturates, I do not believe that the douche is needed in 99 per cent. of our cases. Where the patient has gonorrhœa, of course we *must* douche; but given that she is healthy and that our hands have not introduced any septic matter into the canal, I believe that we shall do harm by douching—in that we needlessly trouble the patient, unless there be retained products of conception.

When sepsis is declared—apart from gonorrhœa—the douche is very useful; but probably the curette is of more service, and indeed often has to be resorted to when the douche has failed to remove retained products of conception.

We now know that the natural secretions of the genital canal make for surgically clean condition, and so long as septic matter is not introduced from without and nothing is allowed to stagnate within, I do not see what, in an ordinary everyday midwifery case, we have to fear.

METHOD OF TREATMENT RECOMMENDED.

So much having been said, it remains for me to indicate a method of treatment which I have found to be satisfactory, and which does not include the binder, the supine decubitus or the "antiseptic" douches.

For some time before term the patient takes iron and ergot, the iron as a hæmatinic and the ergot as a prophylactic of disaster during the puerperium. This if one has the chance of seeing her before labour has set in. Often one's case is only seen when labour has set in, especially amongst Orientals.

When labour has set in the genitals are cleaned. The hairs about the labia are cut short, and the whole pudendum, perinæum and anus washed with turpentine and soap and hot water, the folds of the clitoris and nymphæ being attended to; the inner and anterior surfaces of the thighs are also washed, and a boiled diaper laid under the buttocks. The operator's hands are cleansed with turpentine and hot water and soap. Such instruments as may be required are boiled and then kept in boiled water containing boric acid or phenol. The catheter—soft rubber or gum-elastic—must be kept for hours in a strong solution of phenol, and later in a weaker solution.

For the rare vaginal examinations, no lubricant is used if possible; but, if one be really necessary, boro-glyceride is preferred, as being aseptic and *not* greasy.

When the child is born a large dose of ergot is given, and the placenta is, if necessary, expressed. As soon as the placenta is removed the genitals are thoroughly dried with a pad of absorbent cotton wool, and then a similar pad is applied and kept on with a boiled diaper.

Ergot is given in small doses occasionally for the first thirty-six hours. The patient is kept quiet, but allowed to lie as she pleases, and ordered to get round to the prone position when making water.

As soon as ever she feels inclined for it she is given solid food, and after the tenth day she is allowed to "sit up."

I venture to say that, if my professional brethren in the tropics try this method of treatment—modified to suit the circumstances of the case—they will not be ready to return to the binder and the douche, or to keep their patients lying on their backs, for I have found that what I have above described is easy for the physician, easy for the nurse, and—best of all—easy for the patient.

NOTES ON CASES OF HÆMOGLOBINURIC FEVER IN BRITISH HONDURAS.

By OSBORNE BROWNE, M.B., C.M.EDIN.
Assistant Colonial Surgeon, British Honduras.

THE accompanying notes of cases of blackwater fever in British Honduras may merit a place in the JOURNAL. I have seen three cases since coming to this little place of about 2,000 inhabitants, a year ago. It is worthy of note that two cases were in men who had much to do with cattle, as there has been some talk lately of the analogy between blackwater fever in man and that in cattle, which is known as Texas

fever. One man recovered, but unfortunately I took no notes of his case. There was more jaundice, less loin pain, and no cyanosis with him.

CASE I.—*A rapidly Fatal Case of Hæmoglobinuric Fever.*—A. B., aged 47, Scotch parentage, born in Calcutta, and most of his life spent in the tropics. Kept store and cattle. Often exposed while working with his cattle. Thin, cachectic, strumous scars on neck, and frequently having fever. One day, after coming in from cattle ranch, the urine which he passed was black; then he had a severe chill, felt numbness in arms and legs, and had to go to bed. During the night he had several rigors and attacks of cyanosis. Saw him for first time next day. He was cyanosed and extremely bloodless and cold. The skin was very slightly yellow. There was much bilious vomiting and great thirst. Pulse very feeble, 100. Temperature 102°2'. Pain in the lumbar region, but none in the epigastric region except that caused by the vomiting. The urine was dark-porter coloured. There was no anasarca. He had several rigors and cyanose attacks during the day. Evening temperature 104°0'. He spent a restless night, but appeared somewhat brighter next day. Extremely weak. Though quinine bisulphate tabloids (Burroughs, Wellcome & Co.) were given to gr. 40, the temperature only dropped for a short time to 101°0', and then rose again. Nothing could be retained by the stomach but a little milk, wine and chicken tea. Urine somewhat lighter in colour, but still pain in the lumbar region and indefinite joint pains. Next morning he had another rigor and very severe cyanosis, the temperature rose to 106°0', and in spite of all stimulants he died.

CASE II.—Creole, aged 27, baker, came from Jamaica to Belize in 1891, and from Belize to Stann Creek last March. Tall, loosely built, cachectic-looking man, subject to agues of severe type. Had an attack of fever with red water a year ago in Belize. He says that it was not so severe as this attack, but that he had "much bile on him."

History of present illness. July 22.—At noon I was called. He had had fever since 19th, but got alarmed to-day because he had only passed a small quantity of red water. He was extremely weak, anæmic, and could only whisper. Very restless and anxious-looking. No jaundice. Great pain and weight over stomach, but not much tenderness. No loin pain or tenderness. Much retching up of dark green stuff, and not even a few sips of water could be kept down. Frontal headache. Considerable numbness in arms and legs, but no coldness. Complained of great restlessness and sleeplessness. Tongue had a thick greyish-white fur, red margins and tip. Spleen enlarged to line of navel and liver felt about two fingerbreadths below costal margin. Not costive.

PARASITE OBSERVED IN RED CORPUSCLES.

Examination of blood—small number of a minute, very active body in red cells. It had a central very dark spot, which regularly and gradually appeared, grew darker, then gradually faded away again. Then, after a few moments of complete or nearly complete obliteration, it gradually grew darker again until it became an intense black. It remained an intense

black for a few moments, and then began to grow dimmer once more. This it did with great regularity. As the spot grew dimmer the whole parasite increased in size, and its outline, from being a distinct, regular oval, grew pale, indistinct, somewhat irregular and showed a very faint appearance of four or five dark lines running from its centre to its periphery. Sometimes the dark spot began to reappear at the poles of the parasite, and then suddenly, without any obvious intermediate phase, it became central once more. The parasite is of a yellowish tinge, but grows paler as the spot disappears.

Examination of the urine—Only two ounces was passed before 3 p.m. when a quantity of red water was passed. It was bright red, with no sediment and only a very slight mucous cloud after standing. Much albumen. Later in the day a considerable number of crescents and clear sphere-like bodies were found in the blood.

July 23, 9.30 a.m.—Extremely cold, weak, collapsed, and almost speechless. Still a little green vomit, but not so much retching, and could take a little weak chicken-tea and water acidulated with lime juice. Did not sleep at all, in spite of morphine and acid. hydrocyanic dil. given yesterday. One drachm of quinine was also given during yesterday, and to-day there was no fever. Thirst and restlessness very great. Urine much lighter in colour. Pain and weight in stomach not so great. Much pigment in blood, but no parasites could be found after searching through four slides. Gave him iron, arsenic, strychnine and a little aloes and ordered strong chicken-tea and a little gin.

July 24.—Much stronger. Able to speak naturally. No sleep again last night. Face not so pinched looking. Considerable purging and some green vomit, but he took some cocoanut oil this morning without orders. No fever. Headache and furred tongue as at first. Some full feeling and pain in stomach. Urine lighter in colour. Blood examination nil, except some pigment.

July 25.—Out in hammock, very much stronger, having a chat with a neighbour. Urine still not normal in colour. Ordered port wine and chicken broth frequently, and told him to send if he felt at all worse. He recovered rapidly.

A CASE OF MELÆNA IN CONNECTION WITH QUININE ADMINISTRATION, OBSERVED AT BARRACKPORE, BENGAL.

By R. R. H. MOORE, M.D., Major R.A.M.C.

I RECENTLY read a letter in one of the Medical Journals in which the writer alluded to melæna as a fairly common complication of malarial fever. I have never seen it myself and can only find the slightest allusion to it in the literature of the subject, though I hear that it is observed in other parts of India.

The following very curious case would make me doubtful whether melæna occurring under such circumstances might not be due to the treatment rather than to the disease.

The temperature chart showed the case to be undoubtedly one of enteric fever. Clinically it appeared

to me to differ somewhat from enteric, in that the man was always bright and cheerful, his tongue was never furred, but red and slightly glazed, his abdomen was always flaccid and free from pain or tenderness.

The edge of his spleen could be felt two inches below the costal border. Spots could not be definitely made out on account of the presence of prickly heat.

He was admitted on June 19 and got no medicine until the 24th, the tenth day of the disease. On that morning I ordered him 15 grains of quinine, during the day he passed three stools containing blood. The first was a very large one, consisting apparently of nothing but blood (the man himself states that he was ten minutes passing it and that it amounted to two quarts); the two succeeding stools were small; the blood well clotted in all.

He got no more quinine. He was given dilute sulphuric acid and had no more signs of bowel trouble.

He made a rapid and uninterrupted recovery. That the quinine was the cause of the hæmorrhage in this case I think there can be little doubt.

It is to be remarked that before he got the quinine he was passing a fair number of stools daily, an average of about eight; in character these were in harmony with the diagnosis of enteric fever.

After the quinine the number fell and rarely exceeded two a day.

The fever lasted according to the calculation made—twenty-two days.

On July 24, eighteen days after the subsidence of the fever, Dr. Neild Cook, Public Health Officer of Calcutta, kindly tested his blood for me by Widal's method.

He reported that twenty hours after the application of the test the tendency to clumping was distinct but not strong, and adds, "I should expect this in an early case of typhoid, or in a man who had had his typhoid some time ago."

This contrasted with other cases where the clumping reaction was immediate and strongly marked. There was no history, however, of the man having had enteric before, so the diagnosis of enteric is fairly well established.

RECRUDESCENCE OF PLAGUE IN THE EAST AND ITS RELATIONS TO EUROPE.¹

By PROFESSOR W. J. SIMPSON, M.D., F.R.C.P., D.P.H.

WITH plague in Egypt, on the shores of the Persian Gulf, and now in Portugal, a discussion on the possibility of its further extension will not be out of place at a Congress of the Sanitary Institute. The commonly received view that advancing civilisation caused the disappearance of plague in Europe, and will accordingly prevent a recurrence of plague epidemics, is not supported by historical facts. These show that plague at the end of the 17th century disappeared from the greater part of Western Europe in the course of ten years, and completely in thirty, whilst in the

middle of the 19th century it even more suddenly disappeared from its old haunts in South-eastern Europe, the Levantine countries and Egypt, taking only five years, from 1839 to 1844. No special advance of civilisation characterised these epochs so exceptional in regard to the retrocession of plague. The absence of plague even for a long period is no absolute proof of the immunity of the locality. The city of Bombay was free of plague for nearly two centuries, and yet it would be impossible to state that the city taken as a whole is less protected now by sanitation than it was during the two centuries, or that the people and its government are in a less civilised state.

HISTORY OF THE PROGRESS OF THE PRESENT PLAGUE.

Five years ago, when plague broke out in Hong-Kong, Japan was the only foreign country which, owing to its proximity to the centre of the epidemic, was interested in the outbreak, and showed that interest in an active manner by sending a scientific commission to Hong-Kong to study the nature of the disease. The result of the mission's work was the important discovery by Kitasato of the bacillus or causal agent of plague, which places us in a much more advantageous position of defence against this disease than that of our predecessors. From Hong-Kong the disease spread through Southern China, little note of its extension being made. Two years later it appeared in Bombay, and then the expansive character of the epidemic began to impress itself on the civilised world. In two years it had travelled 3,000 miles westward. In India in the course of six months the epidemic assumed most threatening proportions, the general mortality rising in Bombay city to over 1,600 a week, and important centres in the presidency became infected. Panic, unreasoning resistance to regulations, and flight from the city followed. The infection became still more widely diffused and gradually spread into other presidencies. The disease once having gained a footing in India, continues not merely to spread but also to reappear annually in the infected centres, declining generally during the intense heat and rains and recrudescing in the cooler seasons. The rise and fall is not always regulated by the season; for example, a few weeks ago, Poona was in the throes of her third epidemic, over 150 of her inhabitants dying per day, which in a town of such small dimensions is an enormous mortality, as may be gathered from the statement that if a similar mortality prevailed in London the metropolis would lose over 10,000 persons a day. The mortality in Poona is all the more serious, as I can state, from a personal inspection of the town and of the interior of the houses there, that if inefficient drainage be excepted, which allows of water-logging of the soil, it is in no worse a condition as regards sanitation and overcrowding than hundreds of towns of a similar size in Europe, and is certainly in a much better sanitary condition than many.

Since its advent the plague has caused in India over a quarter of a million deaths. The annual re-appearance, which is a distinguishing feature of the plague in India, is also a conspicuous quality of the

¹ A paper read at the Congress of the Sanitary Institute, August, 1899.

plague in Hong-Kong, and only recently over 100 deaths a week were recorded in the colony.

The European Governments being alarmed at the outbreak in Bombay, the Venice Conference was called to formulate rules and regulations for commerce and pilgrim traffic, having for their object the prevention of the importation of plague, more particularly into Egypt and Europe, as well as into other countries east of the Suez Canal. The rules and regulations have now been in force some two years, and during that time, notwithstanding a most rigid adherence on the part of all concerned to the recommendations of the Convention, plague has reached the Persian Gulf, Penang, the Mauritius, the French island of Reunion, Madagascar, Jedda, Egypt, and if rumour is correct, the Gold Coast of the French possessions in West Africa, and now Portugal, and this extension has happened though the rules were formulated according to the most advanced knowledge on the subject and by the most eminent representatives of the Powers.

Its reappearance in Egypt after an absence of fifty years, with its extension to Portugal, is a matter of profound interest, because it indicates that this plague from China partakes rather of the nature of a pandemic than an epidemic, and possesses that which other plague epidemics for nearly the past 200 years have lacked, viz., the quality of diffusiveness, which defies the precautions hitherto employed against its progress. This is a feature which contrasts strongly with the spontaneous self-limitation of most of the plague epidemics of the 18th and 19th centuries.

THE COMMERCIAL CONDITIONS IN THE WEST ARE FAVOURABLE TO DIFFUSION.

The intimate commercial relations between the countries of the Mediterranean basin are very favourable to the diffusion of plague. It is sometimes stated that there is more danger to Europe from the land route from Bushire, which follows the course of the caravans, than from the sea route from Egypt, but this opinion appears to be based on an insufficient consideration of the intimate commercial relations which now exist between Egypt and the countries of the Mediterranean. When it was a question as to the possibility of plague spreading from India westwards, the condition of the sea traffic between India and Europe rendered it less likely that the disease would spread by sea than by land. The native traders of India and the population of India affected by plague have no direct communication with Egypt or Europe by sea. Hindus are forbidden by the rules of caste to make such a voyage, and Mahomedans come only by sea as far as the Hedjaz for purposes of pilgrimage. The commercial intercourse between India and Europe is maintained by Europeans who belong to a superior class, and who have hitherto enjoyed an exceptional immunity from plague. As regards the native crews that are often employed, distance and medical inspection exclude any slight danger that might be attached to them. Three cases of plague were imported into London in 1896, but that was before it was known that plague prevailed to any great extent in India. The danger to Egypt of infection by plague has not been in its commercial relations with India, but in its religious pilgrimages

which bring its pilgrims in the Hedjaz into very close relationship with Mahomedans from infected centres. But now that plague has arrived in Egypt, a goal has been reached which is favourable to its further extension, because the conditions of sea traffic between Egypt and Europe are very different from those between India and Europe. There is constant communication kept up by fishermen, traders, and travellers, and extending to all classes and including those likely to be affected with the disease. Close proximity also plays an important part.

In the case of a ship coming from India, distance gives time for the development of illness on board, and allows of the adoption of precautionary measures should it turn out to be plague, but a ship coming from Egypt arrives at its destination in the Mediterranean basin before any signs of illness are manifested. Surveillance on board ship on a voyage of over ten days is likely to be superior to surveillance on land, especially when the person coming from an infected country has to pass through several countries. It will be seen, therefore, that the plague in Egypt comes under a set of new conditions which are far from being unfavourable to the westward progress of the disease. Nor is this a matter of mere speculation, for it will be remembered that the cholera in England in 1866 and the cholera in Spain in 1884-1885 followed its prevalence in Egypt.

Further, as a matter of fact, plague has within the last few weeks appeared in Lisbon and Oporto. Portugal being a maritime country and in close connection with Spain, the facilities for extension are considerable, and the proximity of this new centre adds to the danger. From England, Portugal is not more than two or three days' sail, and as there is communication which is not direct with the larger ports where control can be exercised, but with a number of smaller seaports where constant supervision is rendered difficult, the risks to this country are enhanced. The Local Government Board are fully alive to this aspect of the question, as can be seen from their special orders on the subject.

THE BASIS OF PREVENTIVE MEASURES.

Having considered the likelihood of the disease spreading, I shall now turn to the precautions against the disease.

Preventive measures must be based on our knowledge of the disease. Fortunately this has been considerably added to since the plague first broke out in China. Two discoveries of the first magnitude have been made, the one by Kitasato showing that the bacillus is the cause of the disease, the other by Haffkine, proving that from this bacillus a prophylactic may be prepared which has strong protective powers. These discoveries, combined with the fact that plague is a disease which only slowly gains a firm footing in a locality, render the checking or mitigation of an outbreak much more hopeful and certain than before. The recognition of the bacillus permitted observations to be made as to the manner in which the microbe leaves the body. The bacillus has been found in the excreta, urine and sputum. There is a specially dangerous and infective form of the plague, termed the pneumonic, in which the sputum

teems with the bacillus. It is therefore the sputum and excretory discharges to which special attention has to be paid. It is by these that the clothes, bedding and surroundings are likely to become infected.

Precise information of this kind is a considerable advance on that previously known, though it must be admitted that the information does not extend to the life-history of the bacillus in nature, or to the exact methods by which the human body becomes infected. All that can be said at present is that water as a vehicle for the multiplication and spread of the disease plays a very unimportant part. Unlike cholera, plague on reaching a new locality does not break out suddenly into a large epidemic, but extends slowly for some considerable time. A month, and according to Captain Grayfoot, more than six months have frequently elapsed between an imported case and the first indigenous death. The slow progress of the plague in an infected locality, its apparent inability to spread by water, and the mortality which usually occurs among rats previous to a general outbreak, and the fact that the bacillus of the rat plague is identical with the plague bacillus in man, has led some observers to think that the extra-corporeal life of the microbe is in the soil, which is an opinion conforming to the Chinese view that the poison of plague is in the soil. Experiment has so far not confirmed this opinion, but hitherto researches in this direction have not been numerous. A wide gap accordingly exists between what is known and what is necessary to be known to give us control over plague. The danger of extension, as in some other infectious diseases, lies in our ignorance as to the medium agents by which the bacillus gains access to the body, and, if the bacilli are not destroyed immediately they leave the body, the necessary dependence on general rather than on special measures of defence is a defect which may be far-reaching in its results. It is a disadvantage which every opportunity should be taken to remove, and for this purpose it seems to me to be of paramount importance that the Governments of those civilised countries which are affected with plague should not be content in merely endeavouring to combat the disease by every known method at their disposal, such as isolation of the sick, evacuation and disinfection of infected houses, &c., but they should systematically, as a part of the sanitary defence of the country, establish laboratories and special departments for organised research and enquiry into the mode of spread of the disease, for it is only by the adoption of these methods that success is likely to be attained. Fortunately, owing to the researches of Haffkine in another direction, we are in possession of a prophylactic which, if extensively employed, will lend powerful assistance to the other measures which may be introduced to control the disease. An agent which, almost wherever it has been tried, is uniformly successful in reducing the mortality amongst the inoculated when compared with the uninoculated to over 80 per cent., and has reduced the hospital mortality among the inoculated attacked by 50 per cent., is a most valuable weapon of defence which must, if plague continues to increase, come into more and more requisition. That its value is being recognised outside of India may be gathered from the fact that

many applications have already been made for quantities of the prophylactic. Among these may be mentioned the Governments of the Mauritius, Natal, Italy and Russia, the Crown agents for the Gold Coast, the Consul-General of Zanzibar, the Governor of Nicosia, Cyprus, and the Chamber of Mines, Johannesburg.

THE PRECAUTIONS AGAINST IMPORTATION.

To the precautions which were devised by the Venice Conference to prevent importation of plague into a healthy country, and which are embodied in the Venice Convention, I think it is a subject of consideration whether two others might not be added, both depending on exact knowledge obtained and confirmed since the meeting of the Conference. The first is that use should be made of the protective power of Haffkine's prophylactic, and the second is that rats, as they have been proved to suffer from the same plague as human beings and to be agents in its dissemination, should be dealt with at infected ports and on the voyage on ships from infected ports. As regards merchandise, it has always appeared to me that much of the infection is rather connected with the rats which are to be found accidentally associated with grain and other articles of merchandise than with the merchandise itself, and that special precautions should be taken against this mode of extension of the disease. The practical application of Haffkine's prophylactic to the crews of all ships and boats, large and small, coming from infected ports would probably largely reduce the danger of importation of plague, and relieve the anxiety felt at a healthy port when a ship enters having left an infected port a few days previously. Similarly, under certain circumstances and precautions, protection might be given to passengers or travellers desirous of crossing the frontiers into a healthy country.

PRECAUTIONS TO PREVENT SPREAD OF THE DISEASE IN AN INFECTED COUNTRY.

In connection with the checking or stamping out of plague when it breaks out on land, the early notification of diseases is of immense advantage, for it allows measures to be early and promptly applied so that the sick can be isolated, the inmates of the house removed and watched, and the house itself disinfected. For large cities the camping-out system available for villages and small places, and which in these instances has proved so useful, is impracticable, especially if a large area is infected; but in both village and town, whenever a case of plague occurs, not only should the inmates removed from the house be inoculated, but also the inhabitants of the houses within a certain area. By the latter means a zone of comparatively immune persons would occupy what might otherwise become a dangerous focus of disease.

It is possible with proper organisation to deal with an outbreak of plague much in the same way as with an outbreak of smallpox.

Large supplies of Haffkine's prophylactic are necessary in order that every possible contingency may be provided for, and the preparation of these supplies, which require time and a skilled organisation, ought not to be left to the last moment. To meet plague the organisation of defence requires to be placed on as complete and in as efficient a state of prepared-

ness in every respect as the Army and Navy of the country would be if there was danger of invasion.

The sanitary organisation of England inspires confidence that all will be done which our present knowledge suggests to protect the country against disease. But, as stated previously, that knowledge is very imperfect. In the case of an outbreak of disease such as typhoid fever in this country a scientific investigation is undertaken as to its cause and mode of spread. It is known that typhoid fever is caused frequently by contaminated water. This, however, does not prevent further investigation being made in order to ascertain if any new facts can be discovered with reference to the epidemiology of the disease. Epidemic after epidemic is carefully inquired into. A similar inquiry of even a more searching and extensive nature is certainly and most urgently called for in regard to the plague, which is a disease that this generation is not familiar with. The importance to this country of observing its behaviour as modified by conditions in Europe is such that study of it in its epidemiological, prophylactic and curative aspects should be undertaken by Government. To this end it would appear to me advisable that a small commission of experts, of physicians, epidemiologists and bacteriologists, should be sent to the Peninsula for that purpose. It is a matter of Imperial concern and admits of no delay. No expense should be spared, for if the matter is considered from no higher standpoint than a financial one, experience has shown that plague wherever it has acquired a firm hold disorganises and paralyses trade to such an extent that the money losses are immense. The quarter of a million lives lost in India shows the destructiveness of the disease and the importance of making every effort to learn more about the plague with the object of its prevention.

Hitherto I have said nothing as regards general sanitation. That attention to this is necessary goes without saying. While preparing to immunise individuals with Haffkine's prophylactic every endeavour should be made to immunise the locality by sanitary measures. But in this connection it should be borne in mind that excellent water supplies are of little protection against plague, and it has been perhaps to the acquisition of pure water supplies that the attention of sanitarians has of late been chiefly directed, while measures for the dryness and purity of soil have taken but a secondary position, and it is therefore to this branch of sanitation that I would commend that special attention be paid. Localities in which overcrowding, poverty and filth exist have always been the favourite haunts of plague, and it is in these localities in every country and in every large town that protective efforts should be concentrated by the authorities concerned, in order that, if the first line of defence is broken through, the plague may not find conditions favourable to its growth and rapid spread.

Poverty we have always with us; overcrowding may be lessened, or at least regulated or watched, and filth may be easily removed and the dirty spots cleansed.

THERAPEUTICAL NOTES.

FOR NEPHRITIS.—The *Progrès Médical* for June 8 attributes the following to Huchard:—

- R. Tincture of grindelia robusta ... 450 grains.
- Tincture of convallaria majalis ... 150 „
- Tincture of squills... ... 75 „
- M. Fifteen drops to be taken three times daily.

AN APPLICATION FOR URTICARIA.—The *Riforma Médica* for May 9 attributes the following formula to Gaucher:—

- R. Alcohol
- Chloroform
- Sulphuric ether } each ... 3 parts.
- Menthol ... 1 part.
- M. To be applied in the form of spray.

IRON AND CASCARA IN THE TREATMENT OF CHLOROSIS.—The *Riforma Médica* for April 14 gives Liégeois' formula as follows:—

- R. Iron sulphate ... 75 grains.
- Sugar of milk ... 225 „
- Powdered cascara sagrada ... 375 „
- M. Divide into a hundred powders. One to be taken after each meal.

MENTHOL IN THE TREATMENT OF INSECT BITES.—The *Gazette hebdomadaire de Médecine et de Chirurgie* for April 30 gives this formula:—

- R. Menthol ... 1 to 2 parts.
- Sulphuric ether ... 10 „
- M. To be applied lightly with a camel's hair brush. Not to be used near the eye.—*N. Y. Med. Journal*, July 8.

LINIMENT FOR HÆMORRHOIDS.

- R. Extr. hamamelidis fl. }
- Extr. hydrastis fl. } aa ... 3 iv.
- Tinct. benzoini comp. }
- Tinct. belladonnæ ... 5 i.
- Ol. olivæ carbolisat. (5 per cent.) ... 3 i.
- M. Sig. For inunction two or three times a day.—*Adler*.

INSECTS AND DISEASE.

Amât discusses the part played by insects in the spread of disease. The mosquito may carry from place to place the germs of yellow fever and leprosy; bugs have according to Tiktino been responsible for the spread of typhus, the germs of which disease they have taken up when infesting typhus patients. Bugs in the same way have been said to propagate tuberculosis, but this is unlikely. Simond attributes especially to fleas the spread of the plague; these insects attacking first plague infected rats and then passing on to man. Flies, covered as they are with fine dust and pollen may easily convey pathogenic organisms with which they may infect wounds or victuals. The possibility of infection through flies has been demonstrated by actual experiment. Insects may convey micro-organisms either on the surface of their bodies, in their intestine or in the internal organs. Thus flies frequently have tubercle bacilli in their intestinal tract, having obtained them on visits to spittoons. These germs, if deposited on a suitable medium such as milk or broth, would readily multiply. Yersin has found the plague bacillus in the body of a dead fly, and it is obvious that dust containing *débris* of this sort would soon be infected. The part played by the mosquito in the spread of filaria is well known, and allied to this disorder are hæmatochyluria, hæmaturia, elephantiasis, craw-craw and the sleeping sickness. That mosquitos may contain the malaria parasite is well established, though whether it is communicated directly to man by the bite of the animal or whether the contagion is indirect through the agency of water is at present uncertain. Koch has pointed out the analogy between Texas fever and malarial fever and drawn attention to the fact that the former disease is directly transmitted from animal to animal by the agency of tick bites.—*Bull. Gen. de Therap., Janus, April 30, 1899.*

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THE

Journal of Tropical Medicine

SEPTEMBER, 1899.

PLAGUE.

PLAGUE in Oporto brings the disease which started in China more than five years ago very close to our doors. When first made known, the announcement tended to produce much more impression in Europe than the epidemics which continue to ravage certain portions of India. It turns out, however, that the cases in Portugal are few, of a more or less mild character, and irregular in their occurrence. To judge alone from the present behaviour of the outbreak, the conclusion might be come to that the plague is flickering out, and that soon nothing more will be heard of it. Unfortunately this is the more common view, for most people are apt to judge from its present behaviour without reference to past experience, and so the alarm first caused is rapidly subsiding into unconcern. It is easier to imagine that the disease will die out of its own accord than to believe that after 200 years of immunity a plague epidemic at the end of the nineteenth century, with its advanced civilisation, is a possibility. The very important factor is left out of consideration that nearly every plague epidemic behaves precisely in the same manner which is now being observed at Oporto. For instance, it is now known that in Bombay, before

the outburst in October of 1896, there were cases appearing now and again at least six months previously, and in Calcutta unmistakable cases of plague, mild and severe, cropped up eighteen or twelve months before the moderate outbreak of 1898, which was continued into 1899. Similarly in the Great Plague of London cases were observed as far back as November, 1664, though the outbreak did not become serious until May and June of 1665.

The plague at Oporto is accordingly only following the ordinary course that plague normally takes, and there is no justification at present for supposing that the disease has been robbed of its power, or has found the inhabitants immune or exceptionally resistant. It is wiser to read the events in the light of past experience, and to prepare for any contingencies which may happen. If these contingencies do not occur, as was the case at Jedda and other localities where plague spontaneously died out, then all the better for everyone concerned. It should be borne in mind, however, that it is impossible to foretell from the behaviour of the disease in its early stages in a new locality whether an epidemic or not will subsequently follow. In connection with this plague at Oporto it appears that fever with glandular swellings has been prevalent there for some considerable time, following in this respect that which has happened at Hong Kong, Calcutta, Bombay, and Alexandria. In Hong Kong, as shown by Cantlie, there were for one or two years before the outbreak, cases of glandular sickness, occurrences which repeated themselves in the Straits Settlements, and in Calcutta, while recently it is reported that cases of glandular sickness existed in Alexandria as early as January of this year, though plague was not officially declared until May. It is to these cases of glandular sickness which precede the virulent forms of plague that we would like to see investigation directed. Their mildness and prevalence have hitherto only led them to be regarded as curiosities, whereas it is apparent that they are not merely the precursors of virulent plague, but they are also the plague itself, and demand as careful study as the most fatal form. It would seem that it is

to these mild forms of plague that attention must be paid if we are ever to understand the manner in which plague spreads. It is not when plague is epidemic that much will be learned regarding the medium by which the bacillus gains access to the body, or the modes in which the disease spreads, but it is in the early stages, when the cases are few and far between, that enquiry is likely to lead to success; and for this purpose, therefore, it is most important that the glandular sickness should be specially the object of investigation. Commissions of enquiry, to be useful, should be sent at the commencement of an epidemic, and not when the epidemic has fully developed, and the conditions are such that the infection is not easily traceable.

Replies to Articles for Discussion.

THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

I.

SCARLATINA RARE IN INDIA.

WITH reference to Mr. Cantlie's article in the *JOURNAL OF TROPICAL MEDICINE*, July, 1899, on the rarity of scarlatina in the tropics, I would remark the rarity is a fact in India, and especially among natives. In the European army, including women and children in 1897, there were twenty-seven cases of scarlet fever without a death; of these fifteen occurred at Chakrata, a hill station, and six at Muttra in the plains. The cases at Chakrata were said to have been traced to Meerut, but further trace was lost. At two other stations the cases had recently arrived from England. In the *Indian Medical Gazette* for August, 1899, Drs. Cook and Caddie have an article on Scarlatina in India. Measles is fairly common among natives, so is epidemic rose-rash, also dengue now and then appears in returns. On the other hand, chicken-pox and mumps are exceedingly common among natives.

Yours &c.,

W. J. L. BUCHANAN, Major I.M.S.
Bhagalpur, India, August, 1899.

II.

SCARLATINA RARE, ACUTE UNKNOWN; RHEUMATISM AND CHOREA IN BRITISH HONDURAS.

Regarding the prevalence of certain diseases in the tropics, concerning which I see a lot lately in the *Journals*, it may interest you to know that acute rheumatic fever, chorea, valvular heart lesions are unknown here. There are rumours of a few cases of scarlatina years ago in the north of this colony. A large epidemic of measles occurred in the colony a few years ago. A severe epidemic of influenza occurred during 1897. Only a few weeks ago we saw the last of an epidemic of pertussis, which affected nearly every child in the colony, and a few adults. The epidemic extended south to Spanish Honduras and to the neighbouring Cayes, proving fatal to a great number of native children. Typhoid fever does not exist here. If a case of it were to occur, there would be an epidemic to a certainty, owing to the contamination of water supply, which the natives take largely from the river.

Yours very truly,

OSBORNE BROWNE, M.B.Edin.,
*Assistant Colonial Surgeon,
British Honduras.*

III.

NO CROUPOUS PNEUMONIA IN MALTA.

During the six years that the writer was stationed in Malta (1891-6) he does not remember having seen a single case of acute croupous (lobar) pneumonia in which the disease could have been said to have arisen in that place. He had opportunities of seeing and treating large numbers of febrile cases, and was on the lookout for the disease. Numerous instances of catarrhal (lobular) pneumonia occurred in the course of attacks of undulant (Mediterranean) and enteric fevers, &c., but these were, both at the time, in the after course of the disease, and in *post-mortem* appearances, quite distinct from the comparatively short, sharp and acute pneumonia followed by pyrexial crisis so well known in England. On the other hand, he has seen and treated several of such cases transferred from

FRAMBOESIA OR YAWS.

IN different parts of the tropical world an exanthematous disease, contagious in character, ushered in by some fever accompanied by rheumatic pains, and marked by an eruption of tubercles, which become in time moist, fungoid masses, is known by several local names. The Name FRAMBOESIA has been bestowed upon the ailment in consequence of the supposed resemblance of the eruptive masses to a raspberry (*framboise* = a raspberry). The French and Germans designate the disease as *PIAN*; in Ceylon it probably falls under the heading of *PARANGI*; the Fijians term it *Coko*; it is probably also the *PURRA* of the Malays; and some even claim that the "Button Scurvy," which up to the year 1851 was not infrequent in Ireland, belonged to this category.

These names imply a widely spread GEOGRAPHICAL DISTRIBUTION to the disease. It has been recognised in the West Indies, tropical Africa, Ceylon, Java, Fiji and Samoa, and in the Malay Peninsula and Assam.

In Fiji, in tropical Africa and in the West Indies EPIDEMICS of the disease are frequent, and Dr. Daniells states that the Fijians inoculate their children against the disease.

The INCUBATION period is a lengthy one, but owing to the mildness of the initial constitutional disturbance its duration is indefinite. From two to ten weeks of incubation are assigned as the usual limits, but six months even are mentioned as a possible incubatory period. Inoculations have an incubation period of from twelve to twenty days.

The PREMONITORY SYMPTOMS are variable, but usually mild and fleeting; they may, however, occasionally be severe. An initial fever, at one time denied, is in the majority of instances attended by but a slight increase in temperature, but pains in the limbs and a general feeling of being out of sorts are always present.

The characteristic ERUPTION does not appear at once, but changes in the skin gradually lead up to the typical raspberry-like masses from which the disease derives its name. First, furfuraceous patches appear on the skin of the trunk or limbs, or both; the patches may coalesce and give rise to a general furfuraceous desquamation. It frequently happens that this stage may be so slight as to be overlooked. Secondly, papules appear beneath the epidermis, and gradually increasing in bulk push their way up to the surface. Thirdly, the papules now become of the nature of tubercles, consisting of elevated nodules with a yellow top about the size of a pin's head. The yellowness, however, is not due to pus, but to an aggregation of a cheesy material in the apex of the enlargement. At this stage many of the tubercles abort, but the majority rather suddenly increase in size, become fungous in character, pink in colour, and accompanied by a foul-smelling secretion, of a glutinous consistence and a dirty yellow colour. Fourthly, the yaw shrinks, a brown scab takes its place, or an ulcer may succeed, and a stain or cicatrix, according to the depth of the pitting, results.

The face and neck is a favourite site of yaws, and around the mouth and nostrils the tubercles may assume the character of a row or fringe. Even the mucous membrane of the mouth may be affected immediately within the lips.

On the trunk and limbs the eruption is more frequently discrete in character, but around the anus and on the genitals the tubercles may coalesce into groups, ridges or ragged fringes. The sole of the foot may be attacked, and the bursting of the tumour through the thickened epidermis causes considerable pain and swelling. The finger and toe nails are liable to be involved, causing troublesome onychia.

The DURATION of an attack of Yaws varies; it may disappear within two months, but on the other hand recurring crops may continue the disease for twelve months; an average attack lasts three months.

The PROGNOSIS is, in fairly healthy people, favourable; but in persons weakened by any constitutional complaint the joints are apt to become swollen, ulcerated and stiffened; chronic ulcers may form which may assume phagedænic characters; septicæmia may carry off the patient, or he may die of pure asthenia.

The TREATMENT of Framboesia is both general and local. Good food, frequent bathing, and attendance to the sanitary environment of the patient are essential. The local application of disinfectants, either lotions, powders or dressings, is all that is necessary; attempts at destruction of the individual growths are futile. Iodide of potassium or small mercurial doses, or both combined, seem to exercise a beneficial effect upon the eruption.

Hygiene and not inoculation is the keynote of prophylactic treatment.



YAWS.

Photo. of a Hindu suffering from Yaws, contracted in Fiji.

By H. N. JOYNT, M.A., M.D., Fiji.

troopships to military hospitals in the pyrexial stage, or from cargo or passenger ships to the civil seamen's hospital. These latter cases of pneumonia were most characteristic, and their recovery by crisis quite distinct from the continued course and gradual pyrexial fall met with in fever cases in Malta, and only too often followed by relapses.

The connection between ships and cases of croupous pneumonia is an interesting one, which has already been remarked upon. In these particular cases the ships had been some eight to twelve days on the journey from England. Unfortunately the writer does not remember whether cases were landed from ships homeward bound from India or elsewhere. Malta is a damp, chilly place in winter, and though free from snow and ice, there is often much rain and great diurnal variations of temperature. Men get wet through, and frequently remain in their wet clothes, or have even been known to sleep out all night in them without injury. In summer it is hot and dry, except during the damp sirocco wind, while almost every variety of climate may be said to exist during some portion of the year. There is every variety of soil pollution, dirt and overcrowding. There are no rivers, ponds or (in these days) marshes. The soil is very porous sandstone.

It would be interesting and useful to determine the exact etiological factor which interferes with the life-history of the micro-organism and prevents the endemic occurrence of this disease.

M. LOUIS HUGHES,
Capt. R.A.M.C., D.P.H.

PSOROSPERMS IN FISH AND MAN.

The barbel of the Rhone and Saône have for two or three years been decimated by an epidemic of Psorospermia which also affects the tench of the same rivers but without doing them apparent harm. A young man recently entered the Lyons hospital with cavities in his lungs. On examination no tubercle bacilli were found but an abundance of psorosperms identical with those in fishes, and the patient admitted that he ate tench two or three times a week and had taken barbel during his forty-eight hours' sojourn in Lyons. M. Vallin observes (*Revue d'Hygiène*) that such transference has not been before observed, while it is specially interesting in view of recent attempts to establish a relation between certain cancerous growths and sporozoa. It is also remarkable that the same organism should attack beings so diverse as man and the barbel and yet be apparently harmless to the tench. (*Revue Scientifique*, No. 11.)—*Janus*, May.

British Medical Association.

SECTION OF TROPICAL DISEASES.

A DISCUSSION ON PSILOSIS OR SPRUE: ITS RELATIONS (ETIOLOGICAL AND PATHOLOGICAL) TO OTHER FORMS OF TROPICAL DIARRHŒA AND ITS TREATMENT.

Preliminary Remarks.

PERHAPS the most useful and important discussion held at the Section of Tropical Diseases at the recent meeting of the British Medical Association at Portsmouth was that on "Sprue." The Section was particularly fortunate in having present medical men who had gained their experience in the Far East, for it is chiefly in China that typical "sprue" cases are met with; in fact, one result of the discussion seemed to be to throw doubt on the existence of typical "sprue" elsewhere than in China. This may be due to a variety of causes, such as confusion of sprue with various forms of "chronic diarrhœa," "mutatinal diarrhœa," "hill diarrhœa," &c.; but, whatever the cause, the fact seems patent that typical sprue has, up to the present, been recorded only by medical observers in the Far East.

The discussion did not add much to our ætiological knowledge of the disease, but it brought to light a variety in methods of treatment, which is of great interest to the practitioner. Nor were the systems of treatment, if they can be called such, pursued by "quacks" neglected. The home of these irregular practitioners is Shanghai; but the results of their treatment, when fairly weighed, cannot be said to be of so invariably successful a nature as to justify us looking for much guidance in that direction.

I.—GEORGE THIN, M.D., President of the Section.

Description of the Disease.

The disease or diseases which we are to consider are characterised by certain symptoms which are never absent. Irregular and abnormal motions occur in all of them, there being: First, diarrhœa, varying much in severity, frequency, and persistence. Secondly, there are always symptoms of disordered digestion—dyspeptic symptoms; and, thirdly, there is progressive emaciation—symptoms of malnutrition.

Unknown in Great Britain as a Primary Disease.

Looked at more closely, I would invite you to tell us what you know regarding distinct types of disease to which these symptoms are common. As a preliminary it might be worth discussing whether there is any similar disease known to physicians as occurring in this country. This question is worth raising, because two distinguished physicians, of long experience and much consulted for abdominal and dyspeptic complaints in this country, have told me that they had observed cases in persons who had never been out of England. I have not been convinced by this opinion. I have not been able to find in medical literature dealing with diseases in this country an account of any cases answering to psilosis as we know it. My opinion, that the disease is unknown in England, is strongly supported by the fact that one of the most eminent consultants we have ever had insisted that a case of psilosis was a case of secondary syphilis, and another, equally eminent and experienced, informed a patient of mine, who afterwards died of psilosis, that the disease from which he was suffering was chronic malaria, and treated him with full doses of quinine for three months.

Types of Sprue as met with in China and India.

Giving our attention more particularly to this tropical disease, the members will tell us whether they are able to bear me out in distinguishing at all events two distinct types of psilosis. Those who may have done me the honour of reading my book on the subject will know that I divided these cases into at least two categories; one set of cases of which types are common in the Eastern Archipelago, and a

class of cases that are more commonly found in India. In the former, mouth, tongue, and throat symptoms are early prominent, and are even the most pronounced and disagreeable of the symptoms of the disease. In the other, copious watery stools are the chief feature, the mouth and tongue symptoms do not manifest themselves in a decided form until a later stage of the disease, and cases may be met with in which after a duration of a considerable length of time, even years, the tongue and mouth are quite free from specific inflammation, the tongue particularly being still covered with a moist, thin epithelium, and entirely free from localised erythematous and herpetic spots.

In the class to which I have applied the term *psilosis linguæ*, discomfort in swallowing, both in the mouth, throat, and gullet is often very great long before the development of the disease has led to much debility or emaciation. In the Indian type of cases, the diarrhœa alba or white flux type, the mouth symptoms are usually synchronous with advanced malnutrition and emaciation. I have seen cases of this diarrhœa alba or white flux from all parts of the East, including Japan, but chiefly patients from India. The cases associated with *psilosis linguæ* are mostly from the Eastern Archipelago, are not infrequent in Ceylon, and are, proportionately to the other forms, uncommon in most parts of India. These are points on which the members present can give us information.

Chronic Diarrhœa in Persons of Long Residence in the East.

Another form of tropical diarrhœa which is common to most of the Eastern tropical countries of which we have experience cannot well be included in either of these two categories. It is a form of chronic diarrhœa which comes on gradually in elderly persons or in persons of long residence in the East. Its chief characteristic is looseness of the bowels, and, after it has lasted a considerable time, gradual thinning of the epithelial covering and considerable rawness of the tongue occur. It would appear to be due to some degenerative change of the intestinal mucous membrane, although why a degenerative change should show itself more particularly in the intestinal canal is not known. These cases, which well deserve further study, are clinically different from the acute diseases, *psilosis linguæ*, and the *psilosis* which exclusively affects the intestinal canal, and which attacks young persons who may be in good health, the first onset commencing, perhaps, after a very short stay in the tropics.

Sprue Appearing for the First Time in Persons who have returned to Britain after Residence in the Tropics.

Allied to this last group of cases, and perhaps due to a common cause, is the remarkable group in which persons who have been in the East first develop intestinal symptoms long after they have returned to this country. In these cases there are the symptoms common to all the group—diarrhœa, dyspepsia, rawness of the tongue, and emaciation. They are apt to occur in elderly people when the powers begin to fail, although why the mucous membrane of the bowel should be singled out for degenerative change we do not know.

I have seen a sufficient number of these cases to justify my putting them in a separate group. I have recently seen an elderly lady about 65 years of age, who has been home from China for twenty-five years, who never was subject to diarrhœa or intestinal trouble until within the last few months; but the diarrhœa, dyspepsia, and thinly-covered tongue with erythematous points, and the incapacity to swallow anything hot without great pain, differs in no way from the previous group of cases, in which symptoms of a similar kind developed in the tropics in weak persons after long residence.

Pathology.

As regards the pathology of this disease, I cannot say much without repeating the chapter in my book. Our

knowledge must continue to remain very deficient on this subject until we have a record of *post-mortem* changes in persons who have died from other causes while the disease was in its early stages. Before the cases of which we have records came to the dissecting room the whole of the intestinal canal, more particularly the small intestine, had become thin and atrophied. How much of that atrophy is due to a specific cause and how much to a special atrophied condition of the whole body, we do not clearly know. What we do know is that, notwithstanding the general atrophy, there is a special connective tissue development, a true sclerosis in the submucosa, and it has been made out that, at all events in some cases, this sclerotic condition is specially characteristic of the ileum.

The Cause of the Chronic Irritation.

What we are ignorant of is the cause of the chronic irritation that leads to this sclerosis, with its necessary degeneration of the glands and follicles, and why it should specially affect that part of the bowel, and why it should also specially select the œsophagus for special destructive superficial inflammation, as I found it did in one case.

That this cause of irritation may be sufficiently active to produce a real inflammation—a true enteritis—was shown by the intense congestion of the mucous membrane of the ileum and jejunum, particularly of the former, in one of my patients who died from a very acute attack before emaciation had occurred. Any facts that have been observed that can illustrate or throw light on these *post-mortem* conditions will be most welcome. May I take this opportunity of suggesting that, in *post-mortem* examinations in these cases, tissues that are to be of any use must be very carefully hardened, and the *post-mortem* examination should be done within a few hours of the death of the patient. Otherwise the *post-mortem* disintegration of the epithelial layer will be too far advanced to render a subsequent examination of much value.

More particularly in connection with the views of Bertrand and Fontan we shall be glad to have information regarding the existence and nature of any ulceration which may have been found *post mortem*. My own view is that these ulcerations, when present, are secondary in their nature, and that the active agent of the disease may cause a great amount of congestion and subsequent atrophy, with little or no ulceration. I purposely pass over the slight ulcerations found in the rectum and large bowel, which are clearly coincident with symptoms that set in a few weeks of death, and which are mere sequelæ of the disease.

Etiology—Unknown.

The primary cause of this disease is unknown. It is clearly not due to the age of the patient, nor to living in high temperatures. It is associated with residence in certain parts of the world, and is absent in others. That it is of a specific nature is, I consider, made clear by the symptoms of the disease, and whatever it may be, it finds its best soil for development in the ileum. The mouth symptoms may possibly be reflex in their origin. I have seen herpetic eruptions on the soft palate which simulated so exactly ordinary herpes as to suggest a local nerve inflammation. The effects of this specific poison in the small intestine is to alter its secretion and to allow the food bolus to retain its acidity. Hence the dyspepsia, malnutrition and acid stools. It generates a poison which produces extreme anæmia, and it destroys the colouring matter of the bile—the hydrobilirubin. Its activity may be greatly increased by the nature of the intestinal contents, often developing freely in farinaceous and animal foods.

Effects of Diet.

It may be starved out by limiting the patient's diet, and it appears not to grow if the food consists exclusively of milk. An exclusive diet of meat juice seems in some cases to lead to its annihilation, possibly due to the fact that a strict meat-juice diet, to the extent to which it is prac-

ticable, is largely a starvation diet. Milk is not the only nutriment in which it does not thrive. I have previously referred to cases in which strawberries were not only well borne, but were curative in this disease, and possibly other fruits may have similar qualities.

A Milk and Strawberry Diet in Sprue.

I have recently seen a remarkable case, which deserves to be put on record at greater length than my time at present will allow. The patient was a lady under the care of Dr. Playfair (Bromley), with whom I saw her. Her anæmia, wasting, and debility were extreme, and the prognosis I considered bad. She had had the disease for a period of years. Amongst other treatments, milk treatment appeared to have had a thorough trial, and apparently little new was left to suggest. That little consisted in keeping the room night and day at a temperature of 64°, and in never allowing the patient to be removed from under the bed clothes, on the theory that the skin should be kept constantly warm to diminish congestion of the mucous membrane. On this treatment the diarrhœa ceased, but the quantity of milk taken was too small to render recovery possible. After six weeks of this very limited milk diet, at her own instance she began to take strawberries, steadily increasing the quantity. With an increasing amount of strawberries she was able to take more milk, until she ended in taking a large quantity of milk and several pounds of strawberries a day. She made an excellent recovery, became healthy and strong, and can now eat ordinary food without bad consequences. My theory of this and similar cases is that the mechanical qualities of the strawberries have nothing to do with the cure. From that point of view one would indeed consider the small seeds of the strawberries harmful. My view is that, whatever the cause of the disease may be, it does not live on the strawberry juice, and that a diet of strawberries and milk starved it out. If I have made an exception in mentioning this case—for I have purposely avoided going into the treatment—it is only because of the light I consider it throws on the etiology of the disease.

II.—EDWARD HENDERSON, M.D., F.R.C.S. Edin.,
Shanghai, China.

Classification of Cases.

When I arrived in China, in 1868, no distinction was made between cases of chronic diarrhœa and the cases which we now distinguish as cases of psilosis or sprue; when we used the term "tropical diarrhœa" we simply emphasised our belief that the cases we had to deal with were only met with as the result of residence in a hot climate, and were never developed without this as one, and probably the principal, factor in their production. The cases of sprue in which I have been consulted in Shanghai may be roughly divided into two groups:

Group I.—In one the patient has lived in the East for ten, twenty, or more years, and during the whole of that period has enjoyed good, in many cases exceptionally good, health. He suffers now from diarrhœa, which has gradually developed, and is limited to two or three motions in the early morning; the stools are pale, often copious, occasionally sour-smelling and frothy. Latterly he has been troubled by red patches and white spots, which come and go on his tongue and lips, leaving small superficial ulcers behind. He is losing weight, suffers from dyspepsia, and feels weak and ill. Such a case may go on, with periods of improvement almost amounting to recovery and relapses, for years. In the end the patient, with a few exceptions, succumbs directly or indirectly to the disease.

Group II.—The patients composing this group are comparatively young people, and the disease is developed at a much earlier period of residence—from three to six years. In nearly all of the cases there is a previous history of bowel delicacy, shown by repeated attacks of diarrhœa, occurring for the most part during the hot weather, and

insufficiently accounted for by trifling errors in diet, over-fatigue, &c. Sooner or later in one of these attacks the stools assume the sprue character, the mouth becomes sore, and the disease may be considered as established. In nearly all of the cases in this group change of climate was early had recourse to, and the patients, with very few exceptions, completely regained health. In both these groups the leading symptoms are the diarrhœa and the sore mouth; a doctor practising in Shanghai would, I think, hesitate before applying the term psilosis or sprue to a case where the two conditions were not associated.

Pathology.

In the pathology of sprue the chief advance made of late years has, I think, been the limitation of the essential lesion to inflammatory and degenerative disease of the mucous membrane lining the alimentary canal. During the life of the patient the lesions of this membrane are seen in the mouth, and the presence elsewhere is inferred from the diarrhœa and dyspepsia from which the patients suffer more or less throughout their illness. That these lesions are due, in the first instance at least, to a specific poison connected with the development of a hitherto undiscovered bacterium, is of course possible.

Sore Mouth does not Precede the Diarrhœa.

During the progress of a case of sprue I have on several occasions seen the lesions in the mouth distinctly apparent when the stools had become solid as the result of treatment or otherwise, and it is quite common to see the attention of the patient entirely directed to the condition of the mouth. I have, however, no note of any case, nor any recollection of one, in which the sore mouth has preceded the diarrhœa, or has ever had what I could myself regard as a distinctly independent existence. At the same time, in nearly all the carefully-observed cases of which I have preserved notes, these mouth lesions would seem to have developed so early in the history of the case that they could not fairly have been considered as merely the outcome of a condition of general marasmus due to the diarrhœa. Death in a case of uncomplicated sprue is brought about by a process of gradual starvation. At the last *post-mortem* examination which I made in a case of the kind, the diminution in the size and weight of nearly all the organs and tissues of the body was remarkably shown. The liver was greatly reduced in size, but when cut into had to the naked eye a perfectly healthy appearance. The atrophy of the mucous membrane was apparent throughout the whole alimentary canal. It was impossible to say how much of all this was due to starvation and how much to the original disease. My patient had suffered from sore mouth and diarrhœa for at least eighteen months before his death.

Treatment.

Since I returned to England I have been asked what I think of the cures said to have been effected in Shanghai in cases of sprue by the use of drugs the nature and preparation of which has been kept secret by the irregular practitioners who prescribe them.

Irregular Practitioners in Shanghai.

Some years ago there was only one such remedy at all extensively used in Shanghai, and this gradually acquired such a reputation for the cure of diarrhœa, being also credited with the cure of cases of sprue, that the Municipality decided to purchase the secret from its possessor, Mr. Rein. This medicine is now prepared and sold by all the druggists in Shanghai under the name of Rein's Mixture. Rein's Mixture is an infusion of the rough barks of simaruba and cinnamon; the bark of the native (Chinese) cinnamon being the proper bark to use in its preparation. That it is of distinct service in the treatment of chronic diarrhœa which has resisted the usual remedies I can myself testify. As to its value when given in a case of genuine psilosis I have, I confess, serious doubt; the few trials I have wit-

nessed do not encourage me to think that in Rein's Mixture we have got a specific medicine for the treatment of cases of sprue.

At the present day another irregular practitioner has taken the field in Shanghai, and the cures which have gained for him his reputation are attributed to the use of two drugs, one a liquid purgative medicine, and one a white powder having an opposite effect. Nothing is known positively as to the nature or composition of these drugs, but that they are active and in the ways indicated is certain. That patients subjected to this treatment have recovered, and recovered completely, I cannot doubt; that others have received no benefit and have even lost ground while under treatment is equally certain. How many of the cases treated were cases of genuine sprue I have no means of knowing. Personally I am inclined to attribute recoveries of this kind rather to the dieting and general management of the case than to the use of drugs supposed to have a specific action. If I can trust the statements of the patients who have undergone the treatment, the preliminary purging is carried out as a rule much more energetically by the sprue doctor than the average practitioner. The object is attained partly by the strength of the medicine used and partly by repetition of the doses. In advanced sprue any rough handling would be dangerous, but at a comparatively early stage of the disease thorough clearing of the bowel may be of real service. It is to be noted that the use of purgative medicines in full doses is a point insisted on in every system of treatment by drugs which has yet been brought forward.

III.—JAMES WATSON, M.D.

Report of a Case.

A. B., an English lady, went to India in January, 1896. Sometime in that year she suffered from a sharp attack of dysentery, and frequently in other respects her health was far from satisfactory. The hot weather had just set in when distinct symptoms of psilosis manifested themselves. At this time, towards the end of April, she was living at Dehra Dun, a low hill station in the Himalayas. Her first attack of diarrhoea, with the characteristic white, frothy, copious, and offensive stools, occurred at noon on April 31, 1897, and the diarrhoea continued, although she restricted herself to light diet of which milk formed the greater portion. The stools, white, frothy and copious, from the first were not very frequent, occasionally only once in the twenty-four hours. After the diarrhoea had continued for three weeks she removed to a higher hill station in the Himalayas. The diarrhoea was still troublesome; it would however cease sometimes for eight or ten days, but the stools continued to be quite colourless. From the beginning of June, 1897, she had for a period of three months a pure milk diet, that is, milk and nothing else. Under this treatment the diarrhoea ceased, the stools became natural in colour, and the patient thinking she was cured of her malady commenced to take a light, varied, and easily digested diet, and she returned to her home at Dehra Dun. Three weeks after this, early in November, the old symptoms—loose, frothy, white stools—suddenly reappeared. She at once restricted her diet to milk and farinaceous food, and she took regularly thrice daily Hewlett's bismuth and pepsine mixture. This state of affairs continued during November, December, and January, 1898. Towards the end of January the diarrhoea became frequent, weakness increased, nervousness and mental depression marked, and she was strongly advised to leave India for England. This she did one week after her marriage.

She arrived in England in the middle of February, 1898. During the voyage of three weeks, it is interesting to note, there was no diarrhoea, digestion was better, and she arrived in England feeling fairly well—in decidedly better health than when she left India.

For two months after her arrival in England she had very little diarrhoea, but the stools continued to be white,

relaxed, and copious. Towards the end of April she felt the weather to be bitterly cold, and on May 1 she again had recourse to a strict milk diet; but in spite of this she continued to lose strength until she took to her bed in the end of June.

She came under my care on August 10, and was then excessively weak, dyspeptic, anæmic, depressed, and nervous about herself. I at once stopped all drugs, and regulated the amount of milk and the times she was to take it. She continued to take from this time three to four pints of milk daily, divided into doses, which she took every hour and a-half from about 7 a.m. till 10 p.m. The stools, which were white, loose, acid in their reaction to litmus, and frothy, and contained some mucus, soon became nearly solid and of a light golden colour; but every three or four days, without any obvious cause, diarrhoea would supervene. I prescribed, on August 15, one grain of rhubarb and two grains of bicarbonate of soda, which were taken twice daily, and by the end of the month the stools were generally fairly solid, and of a normal appearance as regards colour and consistence.

After a prolonged milk diet of five months and careful nursing my patient was allowed in September small quantities of other food (beef tea, arrowroot, soft-boiled eggs, white fish, &c.). At the first hint of loose stools milk was again prescribed. She gradually gained strength and good spirits, and increased slightly in weight, so that during the months of October, November, and December, she was able to be up and to take ordinary diet, without inducing diarrhoea, and in a quiet way she once more enjoyed life. She was now, therefore, apparently cured of her ailment, though far from strong. She went to San Remo, but the fatigue of the journey induced a relapse soon after her arrival there. Hitherto the tongue had been free from pain; it now became inflamed, raw, and tender, and was the cause of considerable discomfort, sometimes of distress, to her. The strict milk diet was again enforced, and continued till May of this year. During most of the time my patient was in the South of Europe she was confined to bed. On her return to England in the middle of May last she was able to take a moderate amount of ordinary food and some gentle exercise. During the last two months she has gained a very considerable amount of strength and some increase of weight. If, as she sometimes, but rarely, has an attack of diarrhoea, a dose of Gregory powder and milk diet for forty-eight hours puts her bowels in a comfortable and satisfactory condition.

Treatment.

In cases of psilosis I have frequently tried what various drugs will do in their treatment. I regret to say they generally do no good: I am inclined to think they very frequently do harm. I have found, however, rhubarb and soda combined, occasionally small doses of castor oil, and now and then a few grains of Dover's powder, useful. But most cases are best treated without drugs. Prolonged rest in bed and careful dieting are likely to be successful in securing recovery, if these means are used sufficiently early, in the treatment of this disease. I am sure there is no diet more salutary in cases of psilosis than pure milk (boiled or not as the patient likes); and it is wonderful how many patients can be induced to take it continuously for months at a stretch. I am inclined to think that if patients suffering from psilosis are carefully protected by sufficient clothing from chills, they may be as successfully treated in many places in England as in the South of Europe. But as prolonged rest in bed is quite as essential as milk diet, it is necessary that the bedrooms, besides being comfortably warm, should be large and airy and have a southern or western aspect.

IV.—Captain W. J. BUCHANAN, M.B., I.M.S.

Sprue in Natives of India.

It seems to me that it is too generally assumed in textbooks on tropical disease that sprue is mainly a disease of

Europeans residing in tropical countries. It seems to me, however, that the disease is very common among natives of Bengal, and in them often runs a very rapid course. I beg, therefore, to put forward the following statements:—

(1) That primary or protopathic sprue is common among natives of India.

(2) That secondary sprue following on (a) dysentery, (b) acute enterocolitis or enteritis, is common.

(3) That incomplete or arrested sprue is probably very common.

(4) That the condition known as "famine diarrhoea" is essentially the same in its symptoms and ultimate results as sprue.

(5) That in many cases of chronic relapsing dysentery a condition strongly resembling fully-developed psilosis is met with, and that characteristic frothy pultaceous diarrhoea alternates with the dysentery.

Statements (1) and (3) may be taken together. It seems to me that many cases of "chronic diarrhoea" in India are in reality sprue. The history of a "morning diarrhoea" is seldom to be obtained, however. The cases usually begin by complaints of flatulent dyspepsia; this is followed sooner or later by diarrhoea with pale yellow, copious, frothy, fermenting stools; then the diarrhoea may cease for a time but soon recurs; the tongue becomes vesicular, then red and raw; the buccal mucous membrane becomes raw and eroded; very frequently, and often before the psilotic symptoms on tongue, a small ulcerated patch will be found between the last two molar teeth (upper and lower jaw) on one or both sides. This is well known in India, and is called "Crombie's molar ulcer," having been described by Colonel A. Crombie, I.M.S., very accurately some dozen years ago.

Secondary sprue, following an attack of dysentery or acute enteritis, is certainly not uncommon among natives in Bengal. The acute attack passes away, but a chronic diarrhoea remains. In the post-dysenteric form the chronic diarrhoea is at first mainly due to unhealed-up ulceration of the colon left by the previous dysentery. This keeps up increased peristalsis and leads ultimately to atrophy of the glandular functions of the intestines, as is illustrated in the following case:—

Patient had an attack of acute dysentery in March, 1898, remaining in hospital fourteen days. After a few weeks he came back complaining of dyspeptic flatulence, soon ensued diarrhoea with pale, bileless, frothy, pultaceous stools, then appeared the red, raw tongue and inflamed buccal membrane, the molar ulcers. This condition lasted for about six months. He died in October and *post mortem* was found complete atrophy of the glandular structures of the small intestine, and chronic ulceration for about two feet at the sigmoid flexure.

Famine Diarrhoea.

Any one who has read Dr. D. D. Cunningham's description of "famine diarrhoea" in the 1887 report of the Sanitary Commissioner to the Government of India, will recognise the atrophic condition of the intestines therein described as very similar to a case of fully-developed sprue.¹ Again, any one who has had opportunity (as among prisoners) of watching in its whole course a case of chronic relapsing dysentery will recognise in many cases a condition very strongly resembling sprue.² In relapsing dysentery there are intervals in which the dysenteric symptoms are quiescent, when only a chronic diarrhoea remains. Many such cases soon develop into a condition of sprue, as seen in the red-raw tongue and mouth, the peculiar diarrhoea, the anæmia, the dyspepsia, the pearly-white conjunctivæ, the wasting, and great physical and mental debility. The dysenteric symptoms may or may not return, but the patient may linger on for weeks or months, no better and no worse, till either "terminal" dysentery supervenes to carry him

off, or he succumbs to a sudden attack of œdema of the lungs or of pneumonia, his body having been reduced to the last degree of emaciation from a physiological starvation, owing to the almost total atrophy of the glands of the digestive tract.

Treatment.

I believe that many cases, especially among carefully-watched prisoners, are caught in time and cured. For the developed cases the traditional treatment in Bengal jails has been "milk, only milk." This is necessary for natives who won't usually touch meat. For Europeans I prefer the all-meat juice treatment recently advocated by Mr. J. Cantlie in the *Practitioner*. For natives the milk may also be given alkaline (by addition of soda bicarb., potass. bicarb., and common salt, each 6 gr. per pint of milk), or with lime water as used for children. For the preliminary flatulent dyspepsia I know nothing so good as papain (Finkler), or the fresh papaya fruit may be used. But in all cases patience—infinite patience—is necessary.

V.—JAMES CANTLIE, F.R.C.S.

Etiology of Sprue.

In a recent number of the *Practitioner* I ventured to suggest that one possible intestinal irritant in sprue is the acrid vegetable oils used by Chinese (and other native) cooks in the preparation of food.

The disease is so essentially of a nutritive character that it is probable that some irritating substance, or substances, among the ingesta is the cause of the alimentary irritation and disorder. Old tropical hands, in whom the stomacic, hepatic and pancreatic juices flow but in response to stimulation, indulge either in alcohol or strong curries to bring about the physiological effect. The glass of sherry and bitters before food (an Anglo-Indian innovation) is taken for the purpose; but it is not the cause of sprue. Many, if not most, of the persons I have known to suffer from sprue have not only been abstemious, but have been actually total abstainers. Curry, however, is not so easily disposed of. Native cooking generally is open to close criticism as a cause of sprue. Unless prevented from doing so, all native cooks will use vegetable oil instead of butter or dripping, not only in preparing their own food but in that of Europeans. The vegetable oils are cheaper than animal fats, and even when supplied by their European master or mistress with "cooking" butter, the native cook will, if possible, sell it, and supplant it with crude vegetable fats. The acrid, pungent odours of a kitchen where the cook is a Chinaman are proverbial, and it is the chief reason why Europeans insist that the cook-house shall be in a separate building from the dwelling. The vegetable oil chiefly used is derived from rape, and the rape plant, a species of *Brassica*, is largely cultivated in India, China and Japan; the natives of India prefer it to ghee (clarified butter) in the preparation of their hot dishes and curries. Not only so, but rape oil is itself largely subject to adulteration; and hemp oil, rosin oils, and even mineral oils, are added so as to cheapen the production. The Chinese also use the oil expressed from the ground (pea) nut, the oil of the tea-oil plant—a species of *Camellia*—and even the vegetable tallow, derived from the *Stillingia sebifera*, as a substitute for animal fat. The writer believes it is in this direction that the cause of sprue is to be sought, and, without dogmatizing on the subject, would seek to stimulate investigation with this special idea in view.

These vegetable oils, crude and often adulterated, are calculated to ruin any digestion. So astute an observer as Dr. Nansen remarks "that a pemmican made of powdered meat and cocoa-nut oil is a substance scarcely capable of digestion, and that even his very dogs refused it after a time." Cocoa-nut oil is too expensive to use as a substitute for animal fats; but the cheaper oils in use are, especially in their crude form, not one whit more suitable for digestion.

¹ See Manson's article on "Sprue" in Allbutt's "System."

² See Dr. Thin's article on "Psilosis" in Quain's "Dictionary."

Treatment by Meat Diet.

I have for several years now discarded milk. In fact, be the cause of intestinal flux what it may, it is my practice to withhold milk from the dietary. Milk is not a food used by adults in any country, temperate or tropical. Were the liver trained to accommodate itself to deal with a huge quantity of milk from childhood upwards the case might be different; but no adults of the human race use milk as a diet, nor even as the principal part of their diet.

I would at once condemn this method of treatment, and pronounce against it with all vehemence. The effect of giving milk is to bring about a stool which seems more natural because it is nearer the normal in consistence merely; but the stool that is passed on a milk diet is not faecal. Analysis of its constituents shows it to be little more than a mass of soft, cheesy material; the milk curd has been precipitated, and is passed as a compressed mass simulating a solid stool, but in no way is it a normal faecal excretion. There is no faecal odour in the stool, and its pale colour shows that the biliary function is not called into play.

Milk is useful when the object is to keep the patient alive merely, but it does not cure the patient by restoring the organs of the alimentary canal to a healthy state. That state is one of functional inactivity and tissue atrophy. The liver has shrunk to small dimensions, and the disease will continue until the liver is restored to its normal size. But this cannot be done by milk; the liver is not called into play in the digestion of milk but to an infinitesimal degree—not sufficient, in fact, to prevent a retrograde process and atrophy of the organ taking place.

Under an exclusively milk diet the patient may for a time increase in weight and even in strength; but, with or without any accession to the dietary, the relapse comes, and the patient is in no better *visceral* state than before the treatment began.

After considerable experience of the milk treatment, I have given it up as a means of *curing* sprue.

The atonic and physiologically atrophied state of the chylipoietic viscera are, no doubt, at the root of the difficulty as regards the cure of sprue. The liver is reduced in size and in functional activity, and the digestive and absorptive powers of the stomach and intestines are in abeyance. Milk is not the food to call these into existence, and by its administration visceral action is continued in a state of temporary paralyses. Moreover, starchy materials are contra-indicated; they produce diarrhoea and fermentation, and the starch cells are found in the stools undigested. Only by the exhibition of a "meat" diet, in some form, can the action of the liver and other organs be called into play, and it is by the administration of such foods that a *cure* can be effected. Even when the patient is very low—so weak as to be confined to bed—may this *régime* be commended. The patient, if very reduced, must be fed frequently—it may be every quarter of an hour. The diet would then consist of a teaspoonful or a dessertspoonful of beef-juice, made from the fresh beef, not the prepared article; scraped beef, raw meat, beef jelly, calf's-foot jelly, or even plain jelly. As a drink, plain hot water in considerable quantity may be given with benefit. Whey, made by precipitating the curd of milk by rennet, or rice water, made by roasting (not boiling) rice until it is brown and then pouring boiling water over it, are excellent drinks, quenching the thirst and providing nourishment.

As the patient's strength improves, as in all probability it will, the intervals of diet may be increased and raw meat sandwiches given occasionally. Soon finely minced beef, passed thrice or oftener through the mincing machine, beaten up into a thick cream with a little water and with salt added, is to be gradually "warmed" in a saucepan, and given in small quantities, according to the strength of the patient. As the condition improves, a baked apple may be added to the diet and the simplest form of diabetic bread. Subsequently the patient is to be kept on underdone meat,

chicken cream, steamed chicken, game; and, if the diarrhoea is in abeyance, rice, well steamed and dried, is tolerated when the simplest form of pudding will cause diarrhoea or flatulence. Should the patient not be alarmingly feeble, he may be placed on meat, thoroughly minced and carefully "warmed," at once, giving 5 oz. at a time and thrice daily. Between meals the patient is to have beef jelly, chicken jelly, or plain jelly, to satisfy the cravings of appetite, for he must never be allowed to be hungry. When the patient wakes at night, he must be fed systematically.

The very first meal of a "meaty" nature will bring down a bile-stained motion. Pent up so long, there is a tendency for bile to cause looseness, which however, must not induce the practitioner to change the diet or to administer drugs whereby to check the diarrhoea. It is an axiom in the treatment of sprue that astringents, given with the purpose of arresting diarrhoea, are a mistake. When the diarrhoea increases, or the stools show signs of fermentation, the writer invariably administers a teaspoonful or two of castor oil, with the object of sweeping away the offending material, instead of attempting to retain it in the bowel to work further mischief.

The shrunken liver will by this treatment be found to increase rapidly in size, the distension or retraction of the abdomen will subside, but the cure will not be effectual until the liver regains its normal size.

As adjuncts to this system of diet, the patient must be kept in bed, it may be for a few days only. A wet pack, warm at first, but used cold as soon as possible, should be applied to the abdomen night and morning for two hours at a time. The "pack" must be kept firmly in place, so as to exercise some compression, by a large bath towel wrapped round the body, pulled tightly, and secured by safety pins. Between the applications of the wet pack a pad of flannel or cotton-wool, also tightly supported by a body bandage, should be placed over the abdomen.

VI.—CAPTAIN G. LAMB, I.M.S.

Removal of Lime from Milk.

The digestibility of milk, as far as the prevention of the formation of milk curd is concerned, is aided by removing the lime from milk by adding citrate of soda in the proportion of 1 in 400 as recommended by Professor Wright.

VII.—PATRICK MANSON, M.D., LL.D.

Etiology.

Sprue should be regarded not as one disease, but as a variety of diseases with a series of symptoms in common. It is in this respect with sprue as with dysentery or with eczema. They are not single diseases, but rather groups of symptoms common to a variety of diseases. An element in the etiology is the special liability of organs functionally very active to subsequent physiological depression or exhaustion, and their consequent liability to successful parasitic invasion. Sprue as regards the tropics and subtropics should be regarded as pandemic. The earliest description of the disease is that by Hillary, who wrote of it as it occurs in Barbadoes and the West Indies. He had seen cases of the disease from India. Forms of the disease in which a limited portion of the alimentary canal is alone affected do occur. Dr. Manson related a case in which mouth and stomach were alone affected, which recovered temporarily, and later on developed the intestinal form.

Treatment.

There is no exclusive treatment of the disease, and there will not be until we know the etiology. The principles of treatment, however, are the same for all types—physiological rest, rest in bed, warmth, simple diet. A lesson may be taken from the Scotch farmer, who finds that, by ignoring the recognised principles of hygiene, and keeping his cattle in close, warm, badly-ventilated stables, he can fatten them on a smaller quantity of food, and therefore more profitably, than when the cattle are housed in well-

ventilated buildings. The use of fruit, of apples, bananas, and of strawberries is often of great value. Milk is best to begin with, but it must not be too slavishly adhered to.

VIII.—L. W. SAMBON, M.D.

Classification of Cases.

Dr. Sambon said he did not think that the difference observed in the location and extension of the peculiar erosions of sprue along the digestive tract justified the classification of cases into two separate groups, as suggested by Dr. Thin. He attached much importance to the geographical distribution of sprue, which rendered it possible to separate it distinctly from other intestinal disease, such as dysentery and hill diarrhœa, with which it had been confounded. As to the case which occurred for the first time in Europe, but always after residence in China, India, or Ceylon, Dr. Sambon thought an analogy might be found in blackwater fever, enteric fever, and other diseases. The specific germs of those diseases probably became parasitic at an early period, but did not manifest themselves in a typical attack until favourable conditions of low health or metabiosis supervened.

IX.—Major G. M. GILES, I.M.S.

The Hill Diarrhœa of Northern India.

Major Giles remarked that all the cases of true sprue seemed to be described by medical men who gathered their experience in China. His field of observation had been chiefly in Northern India and in Natal, and he could not say that in either country he had ever met with cases of the kind described from China, that is, with early mouth symptoms. Hill diarrhœa, which, held by some to be an analogous disease, was common in Northern India, and he was convinced that a case of the nature described by Dr. Watson would be considered one of hill diarrhœa. If cases of this nature were obstinate they were sent home, and it might be that they did not remain long enough in India to develop mouth symptoms. Hill diarrhœa appeared to be mainly due to suppression of hepatic functions brought about by chills; and, as a general rule, meat diet in any form was harmful, owing, no doubt, to the extra work thrown on the liver by the amount of urea contained in meat. But was this disease sprue? Major Giles stated that his chief object in joining the discussion was to elicit a statement from medical men of Indian experience if they had ever met with a case in India of the exact nature described by observers in China.

X.—SARAT K. MULLICK, M.B., C.M. Edin.

Dr. Mullick gave his experience of cases of sprue which he had met with in this country. He had not found any drug which could be considered a specific. General intestinal antiseptics was the only prophylaxis of any use. He had found milk diet of great benefit. As the case improved Koumiss milk in concentrated form was well borne. He had no experience of the "strawberry treatment." In the next case he would try this most pleasant specific, and, he had no doubt, with the entire concurrence and approval of the patient.

XI.—Dr. FILIPPO RHO, Rome.

Dr. Rho observed that Fontan and other observers stated that they always found small, almost microscopical ulcerations in the last portion of the small intestine and the whole length of the colon. For these reasons they believed that sprue (which they term diarrhœe chronique de Cochinchine) was only a residuum of dysentery, more especially as they believed that traces of blood were always to be found in the stools of patients at the commencement of the disease. The lesions of sprue seemed more like those found in another disease localised in the stomach only, namely, Reichman's disease, which was characterised by dilation of the stomach, sclerosis of its walls, and great increase of secretion of a very fluid mucus (gastro-succorrhœa) which might be com-

pared to the diarrhœa met with in psilosis where the disease was localised in the intestine. Dr. Rho added that he did not mean to imply that sprue and gastro-succorrhœa were identical diseases. He desired merely to call attention to the anatomical analogy.

XII.—Inspector-General TURNBULL, R.N. (Retired).

Dr. Turnbull remarked that he had never met with cases of sprue on board ships of Her Majesty's navy, but he had seen several cases in naval hospitals in England. He had had experience of the meat treatment recommended by Mr. Cantlie and could bear testimony to its efficacy. The treatment by santonin recommended by Dr. Begg, of Hankow, China, did not seem to find advocates in other parts of China, although many medical practitioners had tried it.

XIII.—KENNETH MACLEOD, M.D., Netley.

Professor McLeod said that he had not met with cases in Calcutta corresponding in all particulars with sprue as described by physicians in China. He had often seen cases of hill diarrhœa on their way through Calcutta for England, but he had not observed the characteristic mouth symptoms of sprue in them. It would seem, however, that even in Hong Kong cases of hill diarrhœa existed, for in the *Indian Medical Gazette* some time ago he remembered reference being made to cases of what were termed "matinal" diarrhœa in Hong Kong by Mr. J. Cantlie. It would therefore appear that the two diseases were distinct in type.

REPLY.

Dr. THIN, in reply, remarked that there was no evidence that the liver was diseased in these cases, or that the shrinking depended on anything more than the persistent diarrhœa and general wasting. The white stools were proved by chemical analysis not to consist of undigested milk. Undigested casein had quite a distinctive and different appearance. There was a stage in treatment when milk was no longer required, and when meat was borne well and caused immediate improvement, but that is when the specific diseased condition no longer existed, and when only the sequelæ remained. In the earlier stages, and when the congested condition was active, milk in the great majority of cases answered best, and should, he believed, be always tried in the first instance. After a time—the right time being only found by experiment—meat should be tried. He had tried santonin carefully in two cases without result, and other patients who had consulted him had tried it before he saw them.

A CASE OF PSILOSIS CURED BY STRAWBERRIES AND MILK.

By GEORGE THIN, M.D.

THE case to which I referred at the Portsmouth Meeting, in which strawberries undoubtedly exercised a remarkably curative effect in a case of psilosis, is well worth recording in detail.

MRS. J.—The following summary of the history of the case is compiled from information given me by Dr. Playfair, of Bromley, Kent, and from full notes by the lady herself. The patient, aged 46, whose height is five feet seven-and-a-quarter inches, and whose ordinary weight when in good health is 11 st., after her return to England from Ceylon in 1884 suffered from constantly recurring attacks of sore mouth and diarrhœa. In 1889 she became pregnant after an interval of seven years, and from June to November of that year she was very ill with constant diarrhœa. After her confinement her

life was considered to be in danger, and she tells me that Sir William Broadbent, who was asked to see her, recommended that all medicines should be stopped and that she should have milk alone for a fortnight and not leave her bed during that time. During the fortnight the diarrhoea disappeared, her mouth became well, and she gradually began to take ordinary food. She continued fairly well till 1895, when her health gave way after an illness of her children, and the diarrhoea relapsed. Arsenic and then the santonin treatment were tried without effect, and the loss of weight continued.

In 1897 she again began to take milk diet, and remained in bed for six weeks taking nothing but milk, rising daily during that time for bathing and bedmaking. She was then taken to Brighton, where she was dangerously ill for three weeks. During the rest of that year the symptoms continued with varying severity, the treatment consisting of milk, milk foods, beef-tea, and arsenic.

In November her weight had gone down to 8 st. 2 lbs.

In 1898, from January to March, the weight gradually decreased till it reached 6 st. 2 lbs. She continued to suffer from fluctuating diarrhoea, the diet consisting at this time of beef-tea or turtle soup, Benger's Food, milk—plain, boiled or sterilised—and Koumiss. The diarrhoea increased from April to May, by which time her weight had gone down to 5 st. 10 lbs. After this she was not allowed to be weighed, but she believes her weight still further decreased.

I saw her on May 5, 1898, in consultation with Dr. Playfair, and found her to be extremely feeble, emaciated and anæmic. The prognosis seemed bad. As she had been frequently put on exclusive milk diet there was apparently not much to be hoped from that treatment, but we considered it advisable to try it again with the following precautions. We advised that the temperature of the room should never be allowed to go below 64° F. by night or by day; that no portion of the skin should be exposed for bathing; that she should be washed in bed under flannel; that she should never be allowed out of bed, and that the bed pan should be warmed. In addition she took salol, and, as she was suffering from piles and a bleeding and inflamed rectum, sleeping draughts had to be given.

During this treatment the diarrhoea practically ceased, there being only one and sometimes two motions daily, many of them formed. A few teaspoonfuls of brandy were also given during the day.

The quantity of milk that she was able to take from May 5 till the beginning of June varied from two to three pints daily, and twice during that time she was only able to take a pint and a-half.

When strawberries came in she experienced a strong longing to taste them, and, unknown to her medical attendant, she began to take a few. She commenced taking them in the first week of June, and while they produced no diarrhoea, she was, after she began them, able to increase the quantity of milk to four and four-and-a-half pints a day. She gradually increased the quantity taken, until, on June 28, she took one pound of strawberries and four-and-a-

half pints of milk. Several experiments made with bread and biscuits during this period immediately produced loose fermented motions. She now rapidly increased the quantity of strawberries. On June 30 she took three pounds, on July 2 she increased the quantity to four pounds, and on the 4th to five pounds. Her appetite then rapidly developed, and during this time she took large quantities of milk. As examples of the quantity taken, I note that on July 1 she took seven pints; on July 2 eight-and-a-half pints; on the 3rd and 4th ten pints; on the 5th eight pints, and so on till the 15th, the quantity averaging eight or nine pints a day.

During this period she had, as a rule, one formed motion daily, some days having none.

On July 17 she began ordinary food, with Benger and beef tea, without any bad effect, and steadily increased in weight until she reached nine stones six pounds from the previous record of 5 st. 10 lbs.

She called to see me in the summer of the present year, and looked a healthy, strong, vigorous woman, full of life and energy, without a trace of anæmia. To me it was a veritable resurrection and surprise.

It is well known that there is a fruit cure for psilosis, to which I have referred in my book, where I have also referred to a case related to me by Sir William Gull, in which a lady, who came home from India suffering from this disease, was cured by eating strawberries. I have frequently given fruit of various kinds in treating psilosis, particularly bananas, strawberries, apples, grapes, and, rarely, pears; have sometimes found it well borne and occasionally found it do much good, but I have never with any other fruit obtained such decisive and remarkable results as those related in the case of this patient as following the use of strawberries.

I wish to call attention to the very large quantities taken, and to the fact that the recovery, practically the saving of the woman's life, was due to the fact that when she began strawberries she was able, *pari passu*, to take larger quantities of milk. I wish particularly to emphasise the fact that at a time when bread could not be borne without producing fermentation and diarrhoea, strawberries could be eaten with impunity. I consider that this fact is explained by the assumption that strawberries, like milk, while being nutritious, do not afford the pabulum necessary for the development of the poison that causes the disease. Five pounds of strawberries must contain a considerable amount of sugar, and it is worth considering how far sugar might be administered in the treatment of psilosis. I have tried it on a small scale, but in the light of this case I think it might be pushed. I have never known a relapse produced by sugar.

There is undoubtedly room for active experimental inquiry in the treatment of this disease, and I hope the publication of this case may encourage experiments in the use of highly saccharine fruits, strawberries particularly, and of various kinds of sugar.

Translation.

INTERTROPICAL PATHOLOGY: NOTES UPON
SEVERAL CASES OF FILARIOUS
LYMPHANGITIS.

*Communicated to the National Academy of Medicine of
Rio de Janeiro.*

DR. J. F. DA SILVA LIMA (Corresponding Member).

At the meeting of the Medical Society of Bahia, of May 3, 1889, I read a short treatise which was published in the numbers of the *Gazeta Medica* for that and the following months.

The title of this treatise is "Lymphangitic Fever and its Relations with Filarioid Fever." This title was not mine, as I will afterwards explain, but I adopted it provisionally until new facts should authorise me to propose another more appropriate.

The cases here related are those of two men, very similar under different aspects, but differing in apparent local symptoms. In the first case, which was watched for many years, there were large lymphatic varices, which first appeared upon the groins, and afterwards also in the axillary regions. These varices formed tumours, soft to the touch, yielding to pressure and decreasing in dorsal decubitus; those on the groin were mistaken for herniæ, and the first time that I saw the patient he was even using a double truss. The tumours diminished in a state of cold, and increased during the hyperæmia of the accessions, being then very painful upon the least contact, but with no change in the colour of the covering skin; however, during the more severe attacks longitudinal red streaks were noticed on the thighs and legs; in short, periodical adenitis and lymphangitis, which commenced at the age of seven, recurred at irregular intervals, sometimes even of years. The first attack was accompanied by general convulsions and exterior, visible, local phenomena.

In the other case, more recently observed, there were no local visible symptoms, but the general symptoms were perfectly similar, viz., periodical accessions of fever at irregular intervals, with delirium and other nervous phenomena, and the same convulsions with local and diffused pain.

In neither of these two cases was there chyluria, milky hydrocele, nor lymph scrotum, and but a slight ordinary hydrocele in the first case.

The observation of, and even the spontaneous cure of, the first case (which lasted nearly twenty-five years, some of which were passed in Europe), took place before the discovery of Wircherer's filaria in the blood. Filaria were, however, sought for but not found, at a period long subsequent to the complete disappearance of the accessions of fever, and the lymphangites, and the almost complete disappearance of the varices.

In the second case, however, the filariæ were many times found in the blood by the patient himself, a colleague of ours, and myself.

In my aforesaid work I placed these two cases together because of the great similarity of the general symptoms, and referred in passing to other analogous cases, some previous to the discoveries of Wircherer and Lewis, and some later, in which the filariæ were

not sought for, or in which the fear of the patient prevented the examination of the blood.

It is possible and even probable that in some of these cases, if not in all, especially in the first, filariæ existed in the blood, but this does not amount to more than a mere supposition; and therefore, only mentioning them on account of their analogy with those following, I eliminate them all, only occupying myself with those recently observed, in which the presence of micro-filariæ in the blood were first suspected and afterwards demonstrated.

(1) This case is that related in detail by the patient himself in the short treatise above mentioned, which may be resumed as follows:—Dr. Z. L. F., professor at the Instituto Agrícola, near the town of S. Francisco, on the banks of the river Sergi. He is robust, of a sanguine constitution, and was thirty-four years old when the observation was commenced.

In 1881 he began to suffer from accessions of fever, with lumbar pains sometimes extending up the spine to the shoulders; the pains also radiated towards the spermatic cords, and seemed to extend to the bladder; shivering fits, high fever to 40° C., delirium, and sometimes general convulsions. These attacks, resembling those of miasmatic fever, lasted from six to thirty-six hours, leaving a want of appetite and debility for several days, and recurred at varying intervals of months or weeks until 1884. There was no further attack until 1887. Being consulted in November of that year, I thought first of paludal fever; but examining the case more attentively in January, 1888, I attributed these attacks to deep seated lymphangitis and adenitis, due to the presence of filariæ, not remembering at the time that I had already suggested this idea to my colleague, upon the occasion of a former consultation in 1885. He says that in the first epoch of his sufferings, 1881 to 1884, he successively consulted different doctors, but could never get two opinions which agreed.

Finally, on March 10, 1888, being threatened with another attack, the patient resolved to examine his blood, and discovered live filariæ, which I verified in the slides which he sent me, in which they were already dead. In the night of the 20th, we examined the blood together, and again found them. He continued to make these examinations frequently, until August, and very rarely failed to find the parasites, the few failures being probably due to the high magnifying power of the microscope which he used, 600, 800 diameters and more.

Dr. Z. L. F. informed me that four of his pupils residing in the same school suffered from periodical lymphangitis, and one of them before coming thither found filariæ in his own blood, and that in the locality where the school was built, and even in the building itself, the variety of mosquito commonly called *murissvea*¹ (*Culex pipiens*), or trumpeting mosquito, was very abundant; the professor however only frequented the school in the discharge of his office, residing in the adjoining town of S. Francisco, which is also not exempt from these insects, nor even the house in which he resides.

Having left the said school, Dr. Z. L. F. went to

¹ Which here we call *pernilongo*, or simply mosquito.

the city of Santo Amaro, where he has resided for five years, without suffering more than three mild attacks of fever while he was there; two at intervals of several months, and the third two years ago, which he attributed to paludism, judging himself free from filariæ, which he never again thought it necessary to search for since 1888; but recently, at my instance, he examined his blood two hours before dawn, and contrary to his expectation, after a prolonged examination he wrote to me that he found a live embryo of filaria moving actively.

Therefore this patient has harboured filariæ during ten years, if there has been no re-infection during that period, or for seventeen if it is admitted that the first attacks in 1881 were caused by them.

In his letter my colleague confirms the statement that no exterior signs of lymphangitis or erysipelas was manifested during the attacks, and that there was but a slight infarction of the inguinal glands on the right side for a short time, but so slight that he did not mention it in his notes, and I did not perceive it in my examinations.

The treatment was principally quinine (valerianated), and afterwards thymol, and later oil of mercury, applied in the shape of an ointment rubbed into the thighs and groins. This last remedy twice produced a slight salivation.

(2) V. de C., Bachelor of Laws, magistrate, aged about thirty, resident in the city of Teira de Santa Anna. He consulted me on May 29, 1897. Since February he had suffered more or less severe accessions of fever at intervals of a fortnight, preceded by shivering fits, followed by perspiration, and accompanied by severe pains in the scrotum, spermatic cord, and in the thigh and leg on the same side, without any exterior visible sign of lymphangitis or erysipelas. The attacks lasted two days, and left the patient pulled down. He sometimes brought up dark clots, not knowing whether they were formed of blood or not. Judging, however, that the principal origin at least of the attacks of fever was not incipient tuberculosis of the lungs, in spite of a dry crepitation in the apex of one of them, and considering the symptoms which accompanied the attacks of fever, I told the patient that I suspected the attacks were due to the presence of filariæ, and proposed to him to make a microscopical examination of his blood. However, he was unable to remain in the capital and left for Feira, and never consulted me since. I heard later that he consulted Dr. Alfredo Britto, who obliged me with the following notes, which completed the observation of the case:

"On August 22 of last year (1897), I saw, for the first time, Dr. V. de C. suffering from a severe attack of fever of more than 40° C. preceded by severe shivering, and accompanied by extremely acute abdominal and scrotal pain, great jactitation and delirium. He informed me that these attacks had lately become very frequent, recurring nearly every week, and incapacitating him from the exercise of his profession.

"The resemblance of these attacks to lymphangitis and to erysipelas, with the existence of exterior signs of exanthema, suggested to me that this might be a case of filarious fever, and therefore, when the attack was over, the patient sent me several drops of blood

extracted from the flesh of the finger at dawn, that I might verify the existence of the larvæ which I supposed to be the probable cause of the evil.

"He answered me that the same idea had occurred to Dr. Silva Lima, who had examined him months ago, and that the examination of the blood by a skilful microscopist had yielded no result. I insisted, however, and on the twenty-sixth of the same month I received several slides with the blood still fresh, in which I found different specimens of filariæ still alive.

"My diagnosis being thus confirmed, I prescribed iodoform pilules, quinine wine, and lactophosphate of lime and arsenic. The patient, whom the frequency of the attacks had plunged into a profound physical and moral depression, returned to me some time afterwards, very delighted at their disappearance, to inquire if he should continue the same treatment. I advised him to continue the pilules, to drink sterilised water, and live in a cold, dry climate.

"I must add that this patient informed me that he had suffered more than once from not very abundant hæmoptysis, and not finding any other physical or rational signs to suggest to me the diagnosis of pulmonary tuberculosis, I suspected that the hæmoptysis might also be due to filariæ. I urged him, in case the hæmoptysis should be repeated, to send me some of the blood to see if I could succeed in finding therein Koch's bacillus, or some embryo of nematoda."

(3) A. S., Portuguese merchant, aged forty-five, resident in Bahia from an early age. He suffered four years ago from attacks of fever, at first at intervals of six months, but latterly at intervals of fifteen, twenty, thirty, and sixty days. On the eve of the attack he had lumbar pains which increased during the fever; shivering fits, immediately followed by pains now in one groin and then in the other, extending to the scrotum, and to the thigh and testicle of the same side; at times the pain was felt in the thigh and not in the scrotum, the skin in those regions turning somewhat red and hot; high fever which lasted for hours or a day and a night, and heaviness of the head; the attack left the patient weak for several days. During a year and a half which he spent in Europe he had no attack whatever.

He consulted me on the 28th of last March, a little after one of these attacks, when nothing remained of the local symptoms. Suspecting the existence of filariæ, I enquired if the water-tank of which he made use, together with all the other persons in the house, was often cleaned; and found that the deposit was very rarely cleaned out, and contained abundant impurities, and the water reservoir of his commercial establishment was in the same condition.

The blood was examined by me and other colleagues in the clinical study of Professor Alfredo Britto, who was present. At the first examination no filariæ were found, but upon a second examination, days afterwards, they were discovered alive in the first slide.

This patient did not, and never had suffered, any of the other evils associated with the presence of filariæ.

The treatment, besides adequate hygienic advice,

consisted of quinine, arsenic and kola wine, which was still continued at intervals up to April of the present year.

This patient did not and never had suffered any of the other evils associated with the presence of filariæ.

(4) Z. de A., Portuguese, 22, nine years resident in Bahia. He is employed in the commercial establishment of the preceding patient, and has always resided with him. He has had attacks absolutely similar to those of his master. The first occurred four months ago, and he has had three in all up to the present time.

The first attack commenced by bodily languor, yawning, followed by shivering fits, high fever, heaviness of the head, pain in the left thigh, with redness and heat of the skin; in the second attack the pain extended to both groins and the scrotum, also with redness of the skin; and in the third, which was the most severe of all, he had also orchitis of the right testicle, which continues almost double the size of the left, and slightly painful on pressure, with signs of some liquid in the vaginal tunic.

These symptoms, and the fact that the patient resided with his employer, induced me to examine his blood, when live embryos of filariæ were found on the same day as the first specimen of the employer's blood yielded a negative result.

The blood was extracted from the fingers, according to my instructions, by the patients themselves at six o'clock in the morning, and was examined at eleven o'clock.

The blood of four of Z. de A.'s companions, all young men in perfect health, who had resided in the same house for some years, was also examined, but no embryos of filariæ were found in any. This examination was not repeated as in the case of the master of the house, and therefore it cannot be affirmed that inoffensive filariæ may not have existed in some of his four employes and fellow inmates.

This patient informed me that there were many mosquitoes in the house, and that even in their washing basin it was a common thing to find larvæ of the insect vulgarly called *saltoes*.

The treatment was the same as that prescribed for the employer.

The resemblance of the clinical physiognomy of these four cases is apparent, and the presence in the blood of *Filaria Bancrofti* is a significant fact, which in my opinion cannot be considered as mere coincidence.

Such cases are not, however, very rare, and the natural tendency, which I obeyed in the first case, may have contributed to causing identical cases, in which the local symptoms of lymphangitis are not visible in more or less accentuated form, to be classed as recurring paludal fever, resisting treatment with quinine.

This circumstance justifies the necessity of further careful research into this matter, and it is my resolve to submit it to the enlightened consideration of the Academy, my excuse being that it does not appear that sufficient investigations have been undertaken or published to give us an approximate measure of the relative frequency of the presence of filariæ in the blood of the inhabitants, and whether accompanied by symptoms, and if so, of what nature.

In this sense I am only acquainted with the interesting researches of Drs. Paterson and Hall, here in Bahia, which are to be found in the *Gazeta Medica* for December, 1898. The result of their inquiries is that upon 309 individuals taken haphazard, 169 men and 140 women of different races and ages, who came to the morning¹ consultations of these colleagues for various motives, there were filariæ in the blood of 15, or 1:11½ in the first, and in the second 11 or 1:13, a total of nearly 1:12, or 8.09 per cent.—a proportion very little inferior to that which Manson found in China of 1:10, 8, or 9.25 per cent.

The following interesting facts which seem to have fallen into oblivion result from the patient researches of Drs. Paterson and Hall, namely, of twenty-six filarious patients, only five manifested complaints which might be attributed to filariæ, two suffering from lymph-scrotum, two from evanescent swellings or tumours, which appeared and disappeared over the whole body, and one from hæmaturia, with filariæ in the urine, and not in the blood extracted from the fingers.

It is also to be noted that in one of the women examined who had undoubted elephantiasis in one leg, though her blood was repeatedly examined, not a single embryo of filariæ could be found, although they were sought with the utmost diligence. Among these filarious patients there was a case of albuminuria, followed by jaundice, which did not seem to affect the filariæ at all; and another of a woman with two aneurisms, in whom it is to be noted that microfilaria were most abundant (fourteen in a single slide). Could there be any causal relation between them and the arterial lesions?

(To be continued.)

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

THE BACILLUS OF XEROSIS.—Heiversdorf (von Graefe's *Archives*) draws attention to the difficulty of differentiating accurately the so-called bacillus of Xerosis from the diphtheria bacillus. This would not be a matter of much importance if, as some authors maintain, the xerosis bacillus were seldom found in the healthy conjunctiva; but Heiversdorf declares that it can be detected in no less than 80 per cent. of normal eyes. Hence the importance of possessing a sure means of distinguishing xerosis from diphtheria bacilli. So far, the best method of differentiating them seems to be that described by Neisser in 1897 (vide *Zeitschr. f. Hygiene*, p. 443); this consists in a double staining with methylene blue and "Bismarck" brown, which in a young

¹ The embryos of this filaria (Bancrofti) are rarely found in the blood during the day, and for that reason it was denominated *nocturna* by Dr. Manson to distinguish it from two similar species, one of which was only found in the day (*diurna*), and the other by day and night (*perstans*). It is not unimportant to know that at that time Dr. Hall lived in Dr. Paterson's house, and always commenced his consultations early, even by gas light, and they lasted until half-past six in the morning, as he had no other hours at his disposal in which to attend to the poor, who sought him every day in great numbers. These examinations were continued for two months, and made without distinction from the first five or six patients who presented themselves.

culture (eight to sixteen hours) of diphtheria bacilli brings out a granular structure not seen in cultures of the other bacillus until after the lapse of at least twenty-four hours.

THE BACTERIOLOGY OF EPIDEMIC CATARRHAL CONJUNCTIVITIS.—At the recent meeting of the American Medical Association (June, 1899), Dr. C. A. Veasey, of Philadelphia, read an interesting paper on the "Bacteriology of Acute Catarrhal Conjunctivitis" (the "Pink Eye" of the tropics), in which, after giving a *résumé* of the literature of the subject since the discovery of the Weeks bacillus in 1885, he describes the results of his own examination of sixty-four cases of epidemic conjunctivitis. He comes to the following conclusions:—(1) That for Philadelphia and immediate vicinity by far the most frequent cause of acute catarrhal conjunctivitis is the pneumococcus of Fraenkel. (2) That occasionally it is produced by the Weeks bacillus, and that the clinical manifestations of both are so similar in severe cases that it is practically impossible to distinguish between them without a bacteriological examination. (3) That the experiments of Gifford showing the contagious character of the disease and its reproduction with anaerobic cultures have been fully corroborated. (4) That it has been produced by the writer with a pure aerobic culture. (5) That it is a disease met with more frequently in young adults, but apparently may occur at any age.

THE TREATMENT OF TRACHOMATUS ENTROPION.—The operative treatment of entropion as adapted to the varying necessities of individual cases is probably the most common problem which ophthalmic surgeons in the tropics have to solve. The purposes of a satisfactory entropion operation are to remove the curvature of the tarsus, to supply a new intermarginal space when possible, to weaken the orbicularis, and to fix the skin of the lid so that it cannot glide over the anterior surface of the tarsus. Günsberg (Knapp's *Archives*) believes that the simplified operation of Panas meets these demands. It is performed as follows: Knapp's lid clamp is applied, an incision is made 4 mm. from the lid margin and parallel to it, a section of muscle 2-3 mm. broad is removed and the tarsus exposed, the latter with the conjunctiva is then cut through. Sutures with two needles are then introduced, 3 mm. apart, into the skin of the upper margin of the wound, then deep into the cartilage and out at the upper margin of the tarsal wound, then into the lower portion of the tarsus 1-1½ mm. below the cut edge, passing through its entire thickness and emerging on the mucous side, where they are tried. M. T. YARR.

ITALY.

MALARIA IN ITALY.

In the *Deutsch Med. Woch.*, February 2, p. 69, appears an abstract of Prof. Koch's official report on the scientific expedition to Italy for the purpose of studying malaria. Investigations were made on 120 cases with three necropsies. The summer-autumn form of malaria in Italy is genuine tertian ague, and it is only in the further course of the disease that the fever may change for a time to a quotidian or irregular type. It is not distinguishable from tropical malaria, and the parasites are essentially similar. The so-called crescents, hitherto regarded by many as involution forms, were proved by Romanowsky's method of staining to contain chromatin, and therefore to be capable of life and reproduction. The flagellated forms were proved to act as spermatozoa, and to arise directly from the chromatin bodies, and to consist of chromatin. The commission also found proteosoma in a species of mosquito, and were able to confirm Ross' statements as to the proteosoma being impregnated by the flagellæ in the mosquito's stomach and the subsequent formation of vermicular bodies. Of therapeutic importance is the fact that methylene blue is able to replace quinine in certain cases.

The chief results of the expedition have been to prove the identity of the different forms of malaria, and to lend further support to the mosquito theory.—*Medical and Surgical Review of Reviews*, June.

Medical News.

THE COLONIAL NURSING ASSOCIATION.

Mr. Chamberlain's Speech.

At the Annual Meeting of the Colonial Nursing Association on July 25, the following important speech was made by Mr. Chamberlain:—"I have been asked to move, 'That this meeting approves and confirms the annual report of the Colonial Nursing Association and expresses its cordial appreciation of the manner in which the Executive Committee have given effect to the objects of the Association; and this meeting pledges itself to give its earnest support to the Executive Committee in their endeavour to raise the funds of the Association to £5,000.' The chairman has explained very fully, though in his modesty he says inadequately, the objects of this Association, and on that head he has really left me very little to add. These objects may be expressed in a sentence—they are to provide for our sick fellow-countrymen abroad the same kindly and skilful treatment which all in sickness have at home. And to fulfil that object the Association undertakes the provision of nurses, makes all the arrangements, pays the passages, organises the whole system, gives information, and invites, in effect, a demand upon its time and attention, and, in addition, so far as its limited funds allow, it is prepared, in those districts which either from their poverty or small population are unable to provide the whole of the expense of the nursing for themselves, to contribute something adequate to the purpose. This provision has undoubtedly had most admirable results.

"I see from the report that up to the present time something like forty nurses have been sent abroad, and the reports in all cases are favourable. The demand is increasing, and nothing but want of funds will prevent this Association from completing its work and establishing a similar system in every colony and dependency of the Queen. I am here in my official capacity to testify, as I do most heartily, to the advantages which have already been derived from the labours of this Association, and to the opportunities which are still open to it; and I may say that the Colonial Office is thoroughly satisfied with the experiment which has been tried. We are willing, to the very utmost of our power, to give the co-operation which is needed, and in every way to support the work of this Association. Especially I attach importance to the recommendation which the committee have made, which they speak of as being an ideal—although, like most of their ideals, I think it is extremely practical—that is, that every hospital within our colonies or dependencies which is either supported by the society or by Government funds, should have one or more of these nurses attached to it, who should devote themselves, where circumstances require it, to the service of private patients, and should give the rest of their time to the work of the hospital, which should become their headquarters. That is an ideal which I hope we may realise before very long, and, as I have said, the results of the experiment are thoroughly satisfactory. Almost every day we receive testimonials in favour of the nurses who have been sent out bearing on the importance and value of their work. Only the other day I saw a letter from Colonel Willcocks, who is in command of the West African Frontier Forces on the Niger, in a particularly unhealthy district, in which he paid a most flattering tribute to the virtues of the ladies who had undertaken service with the forces he commands. He declared his belief that the recovery of some of the young officers who had been struck down by that most deadly enemy of the white man, the blackwater fever, was due to the attention and care of the nurses who had been sent out by this Association.

"But I am sure it is not necessary for me to quote testimonials in favour of the value of a work like this, which must commend itself to every responsible and intelligent

person. Nobody can doubt the advantage of skilled nursing in the case of serious illness. Here at home we are endeavouring to secure it in every town and in every village in the country in connection with the District Nurses' Association, and it is universally recognised that the work of the doctor, the surgeon, and the physician is immensely assisted by skilled and sympathetic nursing. But, if it be necessary at home, how much more is it desirable and essential in those tropical colonies where disease is more prevalent, where it is more serious, where the conditions of sickness are more depressing, and where hitherto our sick fellow-countrymen have been left almost entirely to the tender mercies of dirty and indifferent and ignorant natives. Therefore, you will see the field for the operations of such an association is a very large one. The harvest is wide, and in this case the labourers are many, for it is, I think, a testimony to the courage and devotion of British women that we find any number of them are willing, and even eager, to take posts, even in the climates reputed to be most unhealthy, and that the applications for such positions are as numerous as those for countries more favourably situated. All that is wanted is a very modest addition to the funds. As Lord Loch has pointed out, the Association is practically self-supporting. Those who enjoy the benefit of its care and attention are expected, as far as their means will allow, to pay for it, but something like a guarantee fund is required in order that the Association may go forth and continue and complete their work. I cannot doubt that their mission will appeal to everybody who has friends and relations in those distant countries—as, indeed, who has not in the present day? It is a mission to all those who are interested in the development of the great Empire of which we form a part. The importance and the value of the Empire is, perhaps, more justly appreciated now than it ever has been—or, at all events, than it has been in recent times—and with that increased appreciation has come, I am glad to think, a deeper sense of the duties, of the obligations, of the responsibilities which come with it. There has devolved upon this country, partly it may be by the special efforts of some of its citizens, partly by circumstances which appear to be fortuitous, partly on the principle, as I hold, of the process of the natural selection of the fittest—there has devolved upon this country a large share of the control of the tropics, of the administration of vast territories and enormous populations, who will owe peace and security to the influence of Great Britain, which has undertaken the administration of these territories, which is founded, I believe, upon justice and upon the sincere and unselfish desire to secure the progress and advancement of the numberless millions who are committed to our charge.

"If we are pursuing the idea, as I believe we are, in an unselfish spirit, I am perfectly aware also that in its consequences it is of the greatest importance to us, and affects materially our future prosperity and position. Our trade, upon which the very existence of the country depends, tends more and more in the nature of things to become an interchange of products between dissimilar countries, between the temperate countries and the tropical countries, rather than between two or more temperate countries, where the natural advantages and products are the same.

"Accordingly, we have to consider not merely our duty to our race, but our interests, and I say that our success in the work that has been cast upon us, whether in the matter of government or in the matter of trade, whether in administration or in the development of the Empire, depends mainly upon our power continuously to pour into those regions a supply of suitable agents—men, that is, of character, men of capacity, men of an adventurous spirit, who are ready to risk their lives in the cause of civilisation, in the service of the State to which they belong. Fortunately for us, there is no dearth of such men among us. The demand is great, but the supply is always greater, and I think the most confirmed pessimist will find it difficult to despair of his country as long as he sees that these great qualities of

courage, of resolution, of resource, which have distinguished the race in time past and have enabled us to build up this Empire, are still super-eminently present in the men, the explorers and the soldiers, the civil officials, the missionaries, and the traders who carry into those regions the great Pax Britannica, and substitute its blessings for the disturbances and the evils which have been created by barbarous customs of intertribal strife and the cruellest forms of slavery. The lives of these men are one continual struggle, they have to fight more with nature than they have to fight with man, and their deeds, although they may not resound with the publicity which attends great feats of arms in the field, are none the less heroic. Many epics might be written, aye, are written in the records of the Colonial Office, of these modern Ulysses who have encountered greater dangers and have overcome greater obstacles than ever were encountered by their classical prototype. I have desired in these few remarks to bring you to the conclusion I desired to press upon you, that is, that lives such as these, so precious to the Empire, the lives of those who are the successors of those who gained the Empire for us, such lives ought not to be wasted. We owe it to them and to ourselves to do all in our power to preserve them, and to see, so far at all events as that may be possible, that when they are struck down, as unfortunately they are often in the course of their duty, by illness, at all events they shall not want the tending of skilful and kindly hands, and that sympathy, that womanly attention, which will be found to be the best anodyne for their pain and perhaps the most effective cure for their disease. I commend most heartily the work of this Association."

THE LAUGHING PLANT OF ARABIA.

The British and Colonial Druggist states that an Indian medical journal publishes a description of a curious plant which grows in Arabia and in parts of the Western frontier of Hindustan. It is popularly known as the laughing plant on account of the effect produced by eating the seeds. "The plant is of moderate size, with bright yellow flowers and soft velvety seed pods, each of which contains two or three seeds resembling small black beans. The natives of the districts where the plant grows dry these seeds and reduce them to powder. A small dose of this powder has similar effects to those arising from the inhalation of laughing gas. It causes the soberest person to dance, shout, and laugh with the boisterous excitement of a madman, and to rush about cutting the most ridiculous capers for about an hour. At the expiration of this time exhaustion sets in and the excited person falls asleep to awake after several hours with no recollection whatever of his antics."

The Professorship of Pathology at the Calcutta Medical College, hitherto held by the Resident Physician of the College Hospital, in addition to his other duties, is created an independent professorship, as its importance demanded. The Professor will also be the official bacteriologist to the Government of Bengal.

Correspondence.

CLIMATIC BUBOES.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—*Apropos* of Dr. Scheube's articles in your June and July issues on climatic buboes, I find he as well as others have overlooked what I have found to be a rather common cause of non-venereal groin buboes, viz., the irritation produced by scratching patches of that very common complaint in the hot weather, the so-called "dhobie's itch." I made notes over a year ago of at least five such cases in the Central Jail, Midnapur—when, owing to the Calcutta "plague scare," every bubo was looked upon with suspicion.

Yours,

W. J. BUCHANAN, B.A., M.B., Major I.M.S.

Bhagalpur.

Notices of Pamphlets, etc.

THE NORDRACH TREATMENT AT GUIMAR.

In a small pamphlet entitled "A Guide to the New Hospital for the Pure Air Treatment of Tuberculosis at Güimar," Dr. Stanford Harris gives some interesting information about the climate of Güimar on the island of Teneriffe. This health resort, situated on the sunny side of the island some 1,200 feet above the level of the sea, enjoys a wonderfully equable climate, in the winter the temperature seldom going below 50° F. and never below 44°. The atmosphere is dry, the amount of sunshine greater than at the other health resorts, such as Oratava, Santa Cruz or Laguna, and the place is well protected from sudden changes in temperature by the sheltering effect of mountains 7,000 feet high at its back. It is in this region where consumptives have benefited from the dry and sunny climate that a hospital has been built, and is now under the direction of Dr. Harris, who has so fitted it to apply the Nordrach treatment. Fully equipped, this hospital, with the natural advantages which it possesses by reason of an exceptionally good climate, should meet a want and is likely to be much appreciated.

MAICHE'S APPARATUS FOR STERILISING WATER.

Messrs. Maiche, in their endeavour to procure a pure water, have gone further than the ordinary filtration process. They have introduced an apparatus which subjects the drinking water to a temperature of 230° F., but so designed that neither the gases nor the natural inorganic constituents are lost. After this treatment the water is cooled, and supplies a water free of microbes. It is a process that commends itself especially for the colonies and for India, where the difficulty is to meet with unpolluted water.

Communications, Letters, &c., have been received from:—

B.—Surg.-Capt. W. J. Buchanan (Bhagalpur); Dr. R. E. Bennett (Bayswater).

C.—Dr. E. A. Chartres (N.E. India).

F.—Mr. R. Fitch, R.N. (Cape of Good Hope).

G.—Dr. St. Geo. Gray (St. Lucia); Major Giles, I.M.S. (Lydford).

H.—Dr. Hoskyn (Yokohama); Dr. Stanford Harris, Teneriffe; Staff.-Surg. P. B. Handyside (Portsmouth); Dr. Hayward (Birkenhead).

J.—Dr. Henry Joyat (Fiji).

M.—Dr. R. C. MacWatt (Bombay); Dr. R. N. Moffat, P.M.O. (Uganda).

R.—Mr. Sydney Roach, R.N. (West Indies).

S.—Surg.-Capt. W. D. Sutherland (Sangor); Dr. Max F. Simon (Singapore).

T.—Dr. J. C. Thomson (Hong Kong).

W.—Dr. T. D. Wingate (Orlsey).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.

British Medical Journal.
Clinical Journal.
Giornale Medico del R. Esercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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LONDON SCHOOL OF TROPICAL MEDICINE.

By PATRICK MANSON, M.D., F.R.C.P., LL.D.
Physician Seamen's Hospital Society.

THE NEED FOR SPECIAL TRAINING IN TROPICAL DISEASE.

I HAVE been deputed by my colleagues to welcome you to the London School of Tropical Medicine. It is hardly necessary for me to say that we are glad to see you. You are welcome for many reasons, but more especially because you are the first instalment of what we hope will grow in the course of years into a numerous and important band, a band that shall not only leave its mark in the history of tropical medicine, but shall exercise an influence for good in the development of the empire. You are welcome, also, because your presence here is substantial evidence that an idea, long cherished by some of us, has taken firm root in the country, and has at last grown into a vigorous plant, which, we trust, will become by-and-by not only vigorous but also fruitful.

You will find in the introduction to the syllabus which has been placed in your hands a short sketch of the constitution of the school to-day inaugurated, of the reasons why, and of the means by which, it was initiated. It is unnecessary, therefore, for me to enter at length on these matters. The project has been subjected to a good deal of criticism, favourable and unfavourable. One significant and to us satisfactory feature about this criticism is that on the whole the opinions of those best qualified to judge—that is to say, of the men who have themselves done battle with disease in the tropics—has been

distinctly favourable, at all events so far as concerns the desirability, I might say urgency, for such a school. It would be difficult to understand how it could have been otherwise. Many old and experienced tropical practitioners in discussing the subject have bemoaned to me the absence among the medical institutions of their student days of such an institution. I myself, when I recall the years I spent in the East, the many avoidable mistakes in diagnosis and therefore in treatment which I made, and the many opportunities which I missed, cannot but regret that in my younger days I had no training such as will be offered to you here. Many have expressed surprise that, considering the enormous tropical interests of this country, such a school has not been established years ago.

Those about to undertake special work, to do it well, must have special training. Men with tropical experience know very well that there is much that is special in tropical medicine. This being the case, it follows that tropical medicine requires special study; and, because the subject is a very extensive one and is daily becoming more extensive, special teaching and special teachers; and, because the teaching material is to be found in sufficient abundance at only one or two places in the country, special schools at these places.

There are, or there were, some who, while fully recognising the necessity for a school of tropical medicine, have cavilled at the selection of this particular place as its site. Some have said that the Albert Docks Hospital, twenty to twenty-five minutes from Fenchurch Street Station, is too far from the centre of London; others that the clinical material likely to be available is inadequate; others that there are better places elsewhere; others that tropical medicine is already efficiently taught at the military

school at Netley, and that therefore a second school is unnecessary. One and all of these objections have already been fully and satisfactorily met. I have no intention of reopening the subject. Suffice it that the school at the Albert Docks is to-day an accomplished fact; that the student who finds the distance from London too great a tax on his time can be lodged within the walls of the school—a privilege which I am pleased to see has been fully availed of; that the hospital, which, ere another session is over, will be much enlarged, affords a good and sufficient supply of tropical cases, quite enough for much useful practical work; that Netley has not been opened to the public, and were it opened, in consequence of the remoteness from the metropolis it certainly could not and would not be used by many who could and will come to a school in or near London. The objections I have mentioned were carefully considered at the time of the inception of this scheme, and the distinct verdict of those who had worked longest and hardest to carry the scheme into effect was that, *all things considered*, although it may not be in every respect an ideal spot, the Seamen's Hospital at the Albert Docks is by far the most suitable place in the United Kingdom for a school of tropical medicine. However this may be, and whatever may be said or thought about the site, about the curriculum, or about the clinical material, we—that is, my colleagues and myself—have determined to make the best of the opportunities provided for us by the Colonial Office, the Seamen's Hospital Society and the public, in the conviction that if we work hard and earnestly, and pull together, this school will sooner or later prove a great boon to many, and, we believe, play no unimportant part in advancing tropical medicine, and in helping our countrymen to carry out some of the duties imposed by circumstances on our race; that is to say, that it will attain the object for which it was established.

Tropical Medicine a Special Study.

I have said that there is much that is special in tropical medicine. Why should this be? The answer lies at the root of our future studies. To know the cardinal facts of what is special in tropical medicine, to apprehend their bearing, and to be able to give a rational explanation for them is the first, and a most important, step in the science and practice of this department. It has occurred to me, therefore, that a few remarks on one or two aspects of the subject would form a suitable introduction to the course of studies before us.

The principles of pathology, of diagnosis, and of treatment are the same the world over. That is quite true; but it is equally true that the details of tropical pathology, diagnosis, and treatment, and consequently the application of these principles to practice, differ widely from those obtaining in temperate climates. It is with the science and practice of medicine as with the science and practice of agriculture; success or failure in the application of principles depends in great measure on knowledge of detail. A scientific Aberdeenshire agriculturist may be a successful grower of turnips in Aberdeenshire; but without special training and experience I suspect, notwithstanding all his science,

he would be a failure as a coffee-planter in Ceylon. Just so with medicine. It is the knowledge of detail, special experience, special training, in addition to scientific apprehension of principles, that enables the tropical agriculturist to succeed in his special climate at his special work; it is equally the knowledge of detail, the special training and the special experience, *plus* a scientific apprehension of principles, that will enable the tropical practitioner to deal successfully with tropical disease. A physician may be competent to deal with diseases in England but sadly incompetent to deal with disease in Africa.

Etiology a branch of Natural History.

It is well to remember that pathology is, in the main, but the study of a certain fauna and flora—a fauna and flora that inhabit the human body—and the study of the reaction of the human body in the presence of these organisms. In the main, the etiology of disease is but a branch of natural history. Climate, that is, temperature, influences pathology mainly, if not only, inasmuch and so far as it influences the distribution of the pathogenic flora and fauna which, just as in the case of the ordinary fauna and flora, are markedly regulated by atmospheric conditions.

At first sight it is not very evident how pathogenic organisms could be so influenced by any atmospheric condition. The medium in which those pathogenic plants and animals live when they function as disease agencies, that is to say, the human body, is practically the same in all climates. The temperature of the body in health is the same everywhere; it would be hard by chemical analysis to distinguish between the body of a negro and that of a European, and still harder, I opine, to distinguish between the body of an Englishman in India and the body of an Englishman in England. Viewed as culture media for pathogenic organisms, the negro and the Esquimaux are identical, just as are the king and the beggar. Nevertheless it is a fact that the plants and animals haunting the human culture medium in the tropics are in many instances different to those haunting the same medium in temperate climates. Why is this? How can atmospheric temperature influence diversely organisms apparently so uniformly conditioned?

Geographical Distribution of Bacteria.

When you come to look into the geographical distribution of those diseases whose germs reside in the blood or deeper structures—I say deeper structures as opposed to the integuments, which I purposely exclude—you will be struck by the following facts: (1) Except in a very limited class, bacterial diseases are cosmopolitan—that is to say, they can be acquired anywhere or everywhere; (2) diseases of limited geographical range are entozoa. I do not say that all cosmopolitan germ diseases are bacterial, for we know that many entozoa affections also have a cosmopolitan distribution. But I say that all bacterial diseases, so far as we know them, with the exceptions alluded to, are cosmopolitan. Neither do I say that all entozoa diseases have a limited geographical range; not a few are cosmopolitan. But certainly diseases of the blood and deeper tissues, having a limited geographical range, so far as we know them, are

entozoal. It is of the highest importance to the student of tropical medicine, who is a student of what is really a special class of disease having a limited geographical range, that he should at the outset of his studies apprehend the facts on which these two generalisations are founded and understand the reasons for them, for on them depends many of the special features of tropical medicine.

That bacterial diseases should be cosmopolitan is easily understood; the germ passes from host to host without metamorphosis, and practically uninfluenced by the usual media of transmission. It is not killed by a short exposure to ordinary atmospheric conditions. Thus the tubercle bacillus can flourish in every country; plague, leprosy, glanders, influenza, pneumonia, cholera, typhoid and a host of other bacterial diseases, when social and other surrounding conditions are favourable, may be acquired at any time and in any climate.

The only exception to the generalisation propounded lies in those bacterial diseases whose germ is a bacterium which, though usually existing as a saprophyte in culture media having a special range of temperature, may, under certain circumstances, become parasitic. In tropical pathology there is at least one well-authenticated instance of such a bacterium—the micrococcus melitensis, the cause of Mediterranean fever, or, as Hughes calls the disease, febris undulans. There is yet another example from tropical pathology, which, if Sanarelli's recent work is accepted, may also be quoted—the bacillus icteroides, the germ of yellow fever. It is interesting to remark that, whereas the bacterial diseases as a rule are readily and directly communicable, those bacterial diseases which depend upon a saprophytic bacterium are not infectious in the usual acceptance of the word.

Geographical Distribution of Tropical Disease.

In this respect, and as regards their geographical peculiarities, the saprophytic bacterial diseases are a sort of half-way house to the study of the more extensive group of tropical diseases, those caused by animal parasites, of which malaria may be considered the type. The geographical limitations of the animal parasitic diseases are undoubtedly, in many instances, determined by atmospheric temperature. But although high temperature may be an indispensable and ultimate determining factor in their distribution, temperature does not usually operate directly on the causal germ; its operation is usually an indirect one, acting probably through many channels. One of these channels we know.

Most entozoa require at least two hosts, a definitive and an intermediary; without both the germ would die out. Thus in malaria we know, thanks to the recent revolutionising discoveries in which Ross plays so brilliant a part, that a particular genus of mosquito is the necessary definitive host, whilst man, and possibly other mammals, fill the less dignified rôle of intermediary. Recent investigation would suggest the conclusion that without insect definitive and mammalian intermediary there would be no malaria. Consequently, wherever one or the other is absent there is no malaria. Wherever there are no mosquitoes

of the appropriate kind there the disease cannot be acquired. One indispensable biological condition for the mosquito in question is a high atmospheric temperature. This is a principal reason why malaria is a disease more especially of the tropics, but also of the warm season in higher latitudes.

The same or similar explanations hold good for the geographical distribution of those great groups of tropical diseases included under the terms filariasis, dracontiasis, endemic hæmaturia, endemic hæmoptysis, and a crowd of others already known, and probably also of many as yet unrecognised, or, if recognised, etiologically imperfectly understood diseases.

The peculiar distribution, therefore, of a large class of tropical diseases depends, in the first place, on the fact that they are entozoal diseases; in the second place, that the entozoa concerned require intermediary or definitive hosts; and, in the third, that one or other of these hosts requires a high atmospheric temperature—in other words, are natives of warm climates only.

In a limited number of tropical diseases, although an animal intermediary is a necessary or, at all events, a usual agent in transmitting the germ, this animal intermediary plays but a passive rôle. Such is the case in that important disease affecting the horse, ox, and other domestic animals in Africa and India, trypanosoma disease (fly disease, *surra*), which, from an economic point of view, is of such vast importance, especially to the Dark Continent. In fly disease the germ is transferred, as if by an inoculating lancet, on the mandibles of the tsetse fly when this blood-sucking insect, after feeding on a sick, feeds on a healthy animal. Although the fly cannot be said to be a necessity, seeing that its passive office can be discharged by a dozen other agents, yet, as under natural conditions the fly is practically the only transmitting agent, the disease is dependent on this particular insect, at all events in Africa. Now as the tsetse demands tropical conditions, is an inhabitant of tropical countries only, the disease it subserves is necessarily tropical also.

In at least one other tropical disease high temperature acts on the germ in a somewhat different and more direct way. In ankylostomiasis the immature germ, after leaving the human body, in order that it shall become properly qualified for re-entrance into another human host, has to undergo certain developmental changes. As these developmental changes can be effected only in a warm medium, ankylostomiasis is, on this account, necessarily a disease peculiar to warm climates.

I have no time to point out and explain how it is that certain skin diseases are affected in their distribution by atmospheric temperature. Suffice it to say that the parasitic causes of some of them require high temperature and a moist atmosphere in order to flourish on the surface of the body; others, being acquired from plants or animals limited to tropical climates, are necessarily similarly confined.

That the distribution of the germs of the more important diseases is not in any way dependent on the influence of temperature on the human body is proved by the fact that once established in the human body the germ will flourish therein in the coldest as

well as in the warmest climate. Thus, for example, a malaria parasite acquired in tropical Africa may remain alive for months, perhaps for years, after its human host has come to live in England. The same may be said of the filariæ, of the ankylostoma, and of many other parasites; proving that temperature regulates the distribution of disease by directly or indirectly acting on the germ during its passage from human host to human host, and not by any direct influence it may have on that host. This is the first lesson, and a most important one it is, that the student of tropical medicine should learn.

Animals and the Transmission of Disease.

This fact, brought out by these remarks, that the lower animals, especially, I would add, those that are intimately associated with man, play an important part in the transmission of human disease, is only now becoming properly appreciated. In this matter I would point out that, for once in a way, science is vastly in advance of practice. Our sanitarians and the public do not fully recognise all that the community of interest, as regards disease germs, of man and beast means in the spread of disease. At all events if they do understand it they certainly do not act as if they appreciated it.

Plague a Ratborne Disease.

To turn for an illustration to a subject which at the moment is of pressing importance—the extension of plague. This disease, no longer, as it had been for some two hundred years, of academic interest merely, in one of those recrudescences, the explanation of which is still obscure, is again threatening to spread over the world. It is even now knocking at the door of Europe. What have sanitarians done with the mass of fact about this disease, which has been accumulated for them by etiologists and pathologists during recent years; more especially, what have they done with the demonstrated fact that bacillus pestis is a parasite of rats? Judging from the recent history of the disease in Hong Kong, Bombay, Poona, Mauritius, and other places, plague goes on as if there were no such thing as sanitary science. The reason for this, it seems to me, lies in the fact that the sanitarian, like other mortals, is slow to depart from traditions and custom, slow to adopt new ideas, slow to adapt himself to novel circumstances. He has been active and willing enough, but not active in the right direction. Like some physicians, he has only one remedy for every disease. In his efforts at preventing and stamping out plague, he has simply followed old and stereotyped lines applicable to certain other diseases, but not applicable to plague, and has disregarded, practically ignored, the well-attested fact that the ubiquitous rat contracts plague before and more readily than man. From the fact that the rat is attacked by plague before the disease appears in epidemic form in a human community, the inference is indicated that rats are a principal agency in preparing any given place for the epidemic outburst in man; and it also suggests that if the rats in any given place were destroyed, although plague might be introduced, the disease would not spread as an epidemic among

the people; at all events, that its chances of so spreading would be enormously reduced.

I would illustrate what I conceive to be the position of the rat in epidemic plague by a simile. I would compare a plague-threatened, but as yet not invaded, city, to a grate in which the fire is laid all ready for lighting. There is the refractory though combustible coal on top, there is the greasy paper and dry resinous inflammable wood underneath, and there is the lighted match ready to be applied. Drop the match on the top of the coal it flickers for a second and goes out—the coals do not catch fire; but apply it to the paper and sticks underneath; in a moment there is a blaze, the sticks are consumed, the coals catch, and in a little while the fire burns merrily. The coals will now burn by themselves, or if they threaten to go out another stick or two will quickly revive the fire. In my simile the coals stand for the human inhabitants, the sticks for the rodent inhabitants, and the lighted match for the plague germ that has dodged the quarantine intended to protect that threatened city. No sticks, no fire; no rats, no plague epidemic.

As regards communicability, plague cannot be said to be a very infectious disease—certainly not nearly so infectious as small-pox, scarlet fever, or even typhus. The attendants on plague cases, provided they exercise a reasonable and by no means irksome care, do not contract the disease. Man-to-man infection, therefore, cannot be the only, or even the principal, means by which this disease is conveyed; were it so plague would not have spread as it has done in the recent and current epidemics in China, India, and elsewhere. Did its prevalence depend on man-to-man infection, considering the sanitary measures that have been put in operation, sanitary measures which have proved so effective in typhus—a much more communicable disease than plague, it would have died out long ago. There is certainly some way other than man-to-man infection by which plague epidemics are started and kept going. In my opinion that way is the rat. This animal is not included in, and is not affected by, ordinary disinfection operations. It cannot be isolated or segregated like the human being. It runs about from house to house unrestrained, infecting other rats and spreading the disease. It is completely indifferent to Orders in Council, to sanitary authorities, to Acts of Parliament, and even to men in uniform. We know all this; but is the knowledge acted on in that energetic and efficient manner which a lively belief would imply and ensure? Certainly not. Attention in plague epidemics is principally directed against man-to-man infection; hardly at all against the more important rat-to-rat and rat-to-man infection. I believe were rats exterminated in any place, plague could not become established in that place.

We know what the pole-axe policy has done to protect the herds of this country from certain epizootics of a highly infectious nature. The same policy would doubtless be as effective against plague could it be carried out. We cannot as a practical measure, of course, pole-axe human beings in anticipation of plague, but we can kill the rat. It seems reasonable in the light of the facts mentioned to ask why is this

not regularly, and systematically, and efficiently done in every town and village threatened with plague? If you diminish by a little only the chances of survival of a parasite, you will possibly, very probably, extinguish it altogether. No matter how potent and indestructible they may be when once established in the human body, before effecting an entrance parasites are feeble and helpless, and a little matter turns the scale against them; diminish their chances of survival or propagation by this little and you may stop their epidemic diffusion. Therefore, as the rat supplies the best, and most probably the initial, opportunity for bacillus pestis, removal of this opportunity would most likely arrest the threatened epidemic diffusion of plague. Were I asked how I would protect a State from plague, I would certainly answer—exterminate the rats as a first and most important measure; but, in making this recommendation, I would stipulate that the measure be taken in anticipation of the advent of the disease, not when the disease had already shown its presence and the rats are dying by thousands. It is too late then—the mischief has been done. An epidemic in full swing is hard to stop.

I do not know that London may not be again smitten with the plague. Some, relying on the proved efficiency of our measures for the prevention of cholera and on our elaborate sanitation, believe that there is no danger. But I would point out that cholera is not on all fours with plague, and that what applies to the former does not necessarily apply to the latter. There is this important difference between the two, specially important from a sanitary point of view: cholera is a waterborne disease, plague is a ratborne disease. We have taken precautions that our water supply may not be infected with the cholera vibrio; have we taken similar precautions that our rat supply does not become infected with bacillus pestis? Suppose that a single rat infected with plague should escape into the sewers of London from one of the many ships constantly arriving from plague ports. What would be the consequence? The rat would die. Ten chances to one its body, teeming with plague bacilli, would be eaten, as is the habit of rats, by other rats, or the fleas infesting it would transfer themselves to those other rats, and with them plague bacilli. These other rats in turn would become infected with plague. The fat would then be in the fire, and we should have, before very long, an underground epidemic of plague in London. Who can say that the disease would remain underground? At the present juncture, were I the responsible sanitary head of any town in Europe, in anticipation of a possibility, compared to which in horror and in destructiveness a general European war would be a trifle, I would do my utmost to have every rat and, if possible, every mouse in my district promptly exterminated. This done, I should contemplate the approach of plague with equanimity, relying then, but only then, on isolation and disinfection to stop man-to-man infection.

It seems to me that the present measures of plague prevention are like bolting the window against a thief whilst the front door is left wide open; moreover they are exceedingly expensive, exceedingly irksome, exceedingly unpopular, and exceedingly inefficient. Compared to the least of them, the destruction of rats

would be a cheap measure. In the carrying of it out, no one except the rat-catcher need be seriously put about. Efficiently carried out, it would certainly enormously diminish the risk of an introduced plague becoming a pestilence. If ever a sanitary measure was indicated by common sense, it is the destruction of rats in anticipation of threatened plague.

It requires considerable courage to speak of so insignificant a creature as the rat in connection with the diseases and dangers of so dignified a creature as man. Those who think and speak as I have done expose themselves to the railery of the irresponsible smart writer, and such like. I have had some experience of this. I well remember the fun poked at me by the facetious scribe, now many years ago, when I advocated the claims of the mosquito as being a responsible agent in one section of tropical disease; and again, more recently, when I advocated the claims of the same insect as a responsible agent for the propagation of another and more important set of tropical diseases. And now, by advocating the claims of *ridiculus mus*, I fear I run the risk once more of exposing myself to similar gibes. I have my consolation. As regards the mosquito, the laugh is now on my side; as regards the rat, I anticipate the same.

We have high authority for believing that "facts are chieftains that winna ding," and that if we but stick to them they will bring us right in the end. For the prevention of cholera the facts indicate the policy of a pure water supply: for the prevention of malaria the policy of drainage, cultivation, and other methods of mosquito extermination; for the prevention of plague the policy of the rat-catcher. This for these three diseases is my sanitary creed. But, I would emphatically point out, to be effective these measures must be employed in anticipation. When our springs are polluted with cholera vibrios, when the house is full of malaria-charged mosquitos, when the rats are tumbling about the floors drunk with plague, it is too late for general prophylaxis. Then we must fall back on the less desirable measure—personal prophylactics; for cholera the teakettle: for malaria the mosquito net and quinine bottle; for plague Haffkine's injections. I would repeat, to prevent epidemic plague in human communities you must kill the local rats before they are attacked.

The Underlying Principle of Tropical Disease.

In making these remarks about plague, I have wandered somewhat from my subject. In excuse, I would point out that as a hospital we have a special interest in this disease, for when the first line of our national defence against bacillus pestis has been broken through, this hospital, as, indeed, has already happened, is the first medical institution likely to know it; moreover, what I have said accentuates the importance of that great underlying principle of tropical disease, and, indeed, of much disease elsewhere, namely, the interdependence of man and beast in the matter of pathogenic germs. Year by year the progress of medical science makes this more apparent; year by year the scientific study of disease becomes more and more a matter of natural history and less a matter of theory, or, as it often used to be, of metaphysics. The discovery of the bacterial cause

of many diseases concentrated too exclusively for a time the medical mind on that particular class of germ. To-day the protozoon and the helminth, at all events as regards tropical pathology, are in the ascendant. In this school, although the bacterium will not be neglected, necessarily a large share of your time will be occupied with animal parasites, a subject which I fear has not been sufficiently studied hitherto in our medical schools. One of our principal objects will be to make you thoroughly familiar with all the tropical disease germs so far as they are known—bacterial, protozoal, and helminthic; to teach you how to demonstrate these germs, how to grow them, and how to kill them; in other words, how to study them scientifically and practically, that is, from the standpoint of natural history and of practice. We feel assured that this is the only way to arrive at sound methods for the prevention and treatment of the diseases these germs give rise to, as well as for fostering and securing the further advance of tropical medicine.

The Establishment of the School.

Before concluding, I wish to avail myself of the opportunity this occasion affords publicly to thank on behalf of the profession, of my colleagues, and of myself, those who have contributed in any way to the establishment of the London School of Tropical Medicine. To the Secretary of State for the Colonies (Mr. Chamberlain) we are under very great obligations. Without his initiative, and without his sympathy and active support, the hopes of some of us would not have been so speedily and so effectively realised. Apart from philanthropic and scientific considerations, as a piece of practical statesmanship, I confidently predict that the future will prove that of the many public measures Mr. Chamberlain has instituted and advocated this school is by no means the least important or the least promising.

Almost equally indebted are we to the governing body of the Seamen's Hospital Society, who so promptly fell in with Mr. Chamberlain's suggestions and invitation, and who have entered with heart and soul into the enterprise, and who, notwithstanding considerable opposition, have pushed it through to a happy conclusion. They may be sure that the charity they administer will not suffer by being linked to a teaching institution like this. Simply as a matter of financial policy the connection, I venture to say, will turn out to be a wise one for the charity as it certainly is for the school. As a means of forwarding the main object this Society has in view, the healing of the sick, especially of the sick sailor, the connection of school and hospital, to-day commenced, cannot but have a favourable influence.

When the public were appealed to for funds to carry out this scheme, the appeal was promptly and, so far as present wants are concerned, fully responded to. For this generosity we are deeply grateful. In consequence of an almost unexpected liberality we are now in a position to begin work, on a modest scale, it is true, but efficiently furnished with apparatus and free from debt. We look forward, however, to the time when our institution will expand, when our library will grow, our museum prove too extensive

and our classes too large for the present building. Indeed, already we feel that the accommodation at our command is small for our wants. The residential principle seems to be much appreciated; in the near future we anticipate that additional rooms for resident students will have to be provided. A lecture room, too, is needed. To supply these and many minor wants funds will be required; we trust that they will be forthcoming. Although, thanks to the liberality of Sir Henry Burdett, a scholarship of £300 a year, tenable for three years, is in the gift of the school, no provision has been made for up-keep, or for the remuneration of the teachers. The present generation of teachers may be willing to work for nothing; we have no right to assume from this that our successors will be content to work on the same terms. The school requires an endowment. We look to that good fairy the public, which has already been so generous, and for whose generosity we are so deeply grateful, to provide this.

Lastly, I would thank the lay and medical papers, particularly the organ of the British Medical Association, for their valuable advocacy of the school, and for their discriminating and loyal support during a somewhat troubled and anxious period.

Gentlemen, while bidding you a welcome to the school, although feeling assured that we will have something substantial to offer, we would at the commencement of our career, crave a little indulgence. We anticipate that the teaching machinery we have devised will work not quite smoothly at the outset. As the session proceeds doubtless gaps and defects will become apparent. These we trust a little experience will enable us to recognise and rectify. In a short time we hope that all important requirements will be understood and efficiently met; but whatever our initial shortcomings may be, we are confident that by the end of the session you will have learnt at least something worth knowing. And we trust that in after-life, when the experience of actual practice has taught you to appreciate and apply the knowledge acquired within these walls, you will recall with satisfaction, if not with pleasure and gratitude, the few months you are to pass with us, and that you will feel that the time had not been altogether wasted.

A DESCRIPTION OF THE CULICIDÆ EMPLOYED BY MAJOR R. ROSS, I.M.S., IN HIS INVESTIGATIONS ON MALARIA.

By J. M. GILES, M.B., F.R.C.S.

Major I.M.S.

ANY recapitulation of Major Ross' results in the pages of the JOURNAL OF TROPICAL MEDICINE would be quite superfluous, but it may be remembered that the species concerned were distinguished by him as "The Dapple-winged," the "Brindled," and the "Grey Mosquito," and that the "dapple-wing" was the species in which he followed out the life history of an arian hæmatozoon, whose cycle corresponded exactly to that of human malaria, which he had observed previously in Madras, but had failed to obtain a fresh supply of the insect intermediate host.

Major Ross was good enough to give me specimens of each of his species, and as I am at present engaged in collecting all published descriptions of the Culicidæ, I have drawn up descriptions of his species, two of which prove to be new to science, while the third, the "grey," turns out to be *Culex fatigans* Wiedemann, already recorded from the East Indies, and by Wallace from Singapore.

Associated as they must ever remain with Major Ross' epoch-making researches, it appears appropriate that the two new species should be named after him, and, as they belong to distinct genera, is practicable without confusion. The "Dapple-wing" belongs to the genus *Anopheles*, which is distinguished by the fact that the palpi are about as long as the proboscis in both sexes, whereas, in *Culex*, those of the male alone are as long, while those of the female are very short. The three species will therefore stand as, *Anopheles rossii*, *Culex rossii*, and *Culex fatigans*.

The descriptions which follow below are necessarily purely technical, but this is unavoidable, as our readers may rest assured that, if it were practicable to distinguish between species by descriptions couched in popular language, the necessity for technicalities would not have arisen. Moreover, the definitions of the terminology can easily be obtained by reference to any of the numerous introductory works on entomology.

The number of species of Culicidæ already described is so large, exceeding 200 species, that great minuteness is absolutely essential to avoid confusion.

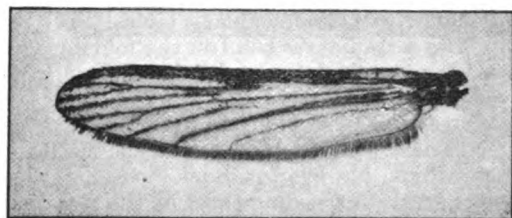


FIG. 1.

ANOPHELES ROSSII.—*Sp. n.*

Size of parts in Male and Female Specimens.

MALE.		FEMALE	
Head	0.5 mm.	Head	0.7 mm.
Thorax	1.2 "	Thorax	1.0 "
Abdomen	2.7 "	Abdomen	3.4 "
Proboscis	2.0 "	Proboscis	1.9 "
Palp	2.0 "	Palp	1.7 "
Antenna	1.8 "	Antenne	1.2 "
Wing	4.0 + 1.0 "	Wing	3.3 + 0.8 "

Wings with four black costal spots, visible to the naked eye, the wing membrane being stained as well as the scales; anterior femora not dilated at their point of attachment; the palpi basally pale branched, in both sexes on most of the joints; general coloration rather pale grey, with the abdomen centrally white spotted.

Male.—Head covered with a mixture of brown and whitish scales, the latter preponderating on the vertex, the nape nearly black. Eyes black, with a narrow whitish margin; antennæ pale fulvous, with silky brown verticils, the last joint rather darker at the tip, the

basal entirely golden brown. Proboscis nearly black, golden brown at the tip. Palpi dark brown, the distal joint pale fulvous at the tip and externally; a basal band of the same colour on all but the basal joint; the last joint is expanded into a broad, compressed club. Thorax of the same tint as the head, with a minute blackish shoulder patch, and some indistinct darker markings laterally, and on the coxæ. Legs uniformly pale grey, rather darker externally, and on the last two tarsal joints the claws are small, and without basal teeth.

Wings longer than the abdomen, hyaline, the veins with white scales except on the spots and dapplings; there are four distinct black costal spots, the largest of which is placed opposite the origin of the second longitudinal vein; close behind it is one of smaller size; at the tip of the wing is the smallest of the series, and the fourth is placed midway between this and the large spot first mentioned. Besides these spots, visible to the naked eye, many of the veins are black for small portions of their length, the most noticeable being the 4th long., which is black from its forking to the transverse veins; the tips of the 4th, 5th, and 6th longitudinals; at the root of the 5th long., and a minute spot on the costal at its base. The numeral transverse vein is curved, and placed very close to the root of the wing, so that it is with difficulty made out; the subcostal transverse is placed a good deal beyond the middle of the auxiliary vein, and quite near the origin of the second longitudinal, the latter being placed much nearer to it than to the origin of the supernumerary transverse; auxiliary vein reaching the costal before the level of the tip of the hinder branch of the 5th longitudinal; 3rd longitudinal appearing at the junction of the supernumerary and middle transverse veins, which together form a continuous line, joining the 2nd and 4th longitudinals, and a little beyond the tip of the 6th longitudinal, all these cross veins being of about equal length, but the numeral transverse is hard to make out, being placed very near the base of the wing; first submarginal, slightly longer and narrower than the second posterior cell, the base of the former slightly in advance of that of the latter; base of the anal cell a little beyond the level of the origin of the 2nd longitudinal vein.

Halteres with white stem and black club.

Abdomen light grey with a narrow darker hinder border to the segments, the two posteriors of which are distinctly darker than the rest; there are some ill-defined darker lateral striæ, and a dark median spot on the two proximal segments; the claspers are provided with very long claws.

Venter uniformly pale brown, except for a pair of lateral white spots, separated by a darker median line on the four hinder segments.

Female.—Closely resembles the male. Her palpi are uniformly black, except the apical half of the end joint, and a band at its base and another on that of the penultimate, which are more or less golden brown. The antennæ are black throughout, with scanty verticils of lighter tint; the basal joint golden brown. The legs have the coxæ pale drab; the femora very pale at the base, darker at the distal end, where there is an external black line, which is continued on the tibia,

with the interruption of a faint lighter knee spot; the hind legs show four lighter basal bands to the joints. The venter of the abdomen is pale drab, with indistinct darker lateral lines, and a dark hinder border to the three or four distal segments.

Habitat.—Calcutta, India. Collected by Major Ross, I.M.S., and found by him to be the intermediate host of the parasite of a form of avian malaria.

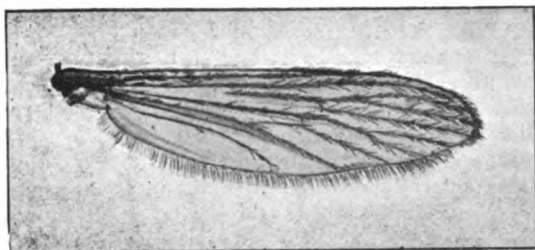


FIG. 2.

CULEX ROSSII.—*Sp. n.*

Size of parts in Male and Female Specimens.					
MALE.			FEMALE.		
Head	0.6 mm.		Head	0.8 mm.	
Thorax	1.3 "		Thorax	1.5 "	
Abdomen	2.8 "		Abdomen	2.9 "	
Proboscis	2.2 "		Proboscis	2.1 "	
Palpus	2.2 "		Palpus	0.45 "	
Antenna	1.3 "				
Wing	2.9 + 0.7 "		Wing	3.4 + 0.9 "	

Wings unspotted. Tarsi white-ringed at the bases of the first and second joints only on all the legs, which also show minute knee spots. Body dark brown, brindled with white in the male; the abdomen with basal white bands to the first five segments, and a median white line: coloration of the female, lighter and less brilliant, the brown being lighter and the white less pure.

Head black, with a white nuchal collar. Eyes black, with brown reflexes and a margin of white scales. Antennæ, with the globular basal joint black, saving a broad ring of white on its anterior face, round the insertion of the second joint; the remaining joints whitish, with black verticils, the roots of which have the effect of minute black bands; the last two joints deep brown, and together forming more than a fourth of the length of the appendage. Proboscis deep brown, a little paler beneath, near the end. Palpi exactly equal in length to the proboscis, black with broad basal white bands, those of the first two joints being especially broad and distinct; the basal joint short, the second forming more than half the entire length of the appendage, the last two of equal length. Thorax (when denuded) dark brown. In some specimens, there are traces of white scales, so that, in all probability, there are white markings in unrubbed specimens. There are also white spots on the sides, corresponding to the white marks on the coxæ, and also beneath the insertion of the wings. Scutellum dark brown, glabrous, or denuded.

Legs differing comparatively little in length, those of one specimen being 6.1, 6.5, and 7.5 mm. for the fore, middle and hind pairs respectively; brindled with light and dark brown, with the coxæ covered with pure white scales externally; the femora paler

at the base, especially internally, where they are nearly white, with a minute white knee spot, and an inconspicuous basal white band to the two first tarsal joints on all the legs; the remaining tarsal joints being unbanded. The fore and hind claws are each provided with a well-marked basal tooth, those of the fore leg being, however, much the larger: the claw of the middle leg is untoothed, but is otherwise as large as that of the fore.

Wings hyaline, with dark scales; auxiliary, joining the costal opposite the supernumerary transverse vein, and considerably before the level of the tip of the fifth longitudinal; supernumerary and middle transverse veins at the same level. Subcostal transverse, much nearer the origin of the second longitudinal than to the numeral transverse; posterior, placed more than twice its length behind the middle transverse, and of about equal length, the former joining the anterior branch of the fifth longitudinal a good deal before its middle; anterior branch of the fifth longitudinal originating considerably before the tip of the sixth, and its tip joining the posterior border well beyond the base of the second posterior cell, which latter is considerably shorter than the first submarginal, although much wider. Halteres pale-tinted.

Abdomen black, with a median white line, broadening at the front of each of the first five segments, into a distinct, broad band, and with a barely perceptible white fringe to the hinder border. The sixth segment is brindled with white and black scales in the middle, and has a white spot on either side; the seventh is entirely black, and there is a minute terminal white spot on the last. Ventrally, the white forms the ground colour, with sinuous black lateral lines, which meet to form a transverse band on the fourth segment. The last segment is armed with exceptionally large, incurved claspers.

The above applies specially to the male; the female is larger in nearly all dimensions, except the length of abdomen, which, however, is much stouter, the thorax and wings greatly exceeding those of the male in size. The antennæ are black throughout, and appear proportionally longer than those of the male. The palpi are also black and without bands; the two proximal joints shorter than the distal, being together barely equal to one of the latter, which are subequal. The general coloration of the female is altogether less brilliant, the dark parts being much paler, and the whites impure. The dorsum of the abdomen is dark greyish brown, with an ill-marked light basal band to each segment, and ventrally, the brown lateral lines are joined by cross bands on nearly every segment, the white parts, external to the sinuous dark line, giving the appearance of a series of lateral light spots.

Habitat.—India (Calcutta). Major Ross, I.M.S.

Note.—This species closely resembles, but is not I think, identical with *C. tæniatus*, described by Wiedemann, from Savannah. The dorsum of the thorax is so denuded of its scales in all my available specimens, that the characteristic adornment of the female of Wiedemann's species, which is specifically stated to be noticeable in only unrubbed specimens, would not be in any case discernible. These markings were, however, darker than the grounding, while there are sufficient remains to show that the adornment of the present species was white on a dark ground, though not to enable one to judge its exact character. On the other hand, I can make out no trace of the snowy scutellum of *C. tæniatus*, and though the abdomen of

several of my specimens is in very fair condition, it does not correspond to the description of that species.

The palpi of the male correspond, but those of the female are black, or at most, show only a trace of white at the base of the terminal joint, instead of being white at the base and tip. For these reasons, without laying undue stress on the American habitat of *C. tæniatus*, it appears more probable that they are distinct species.

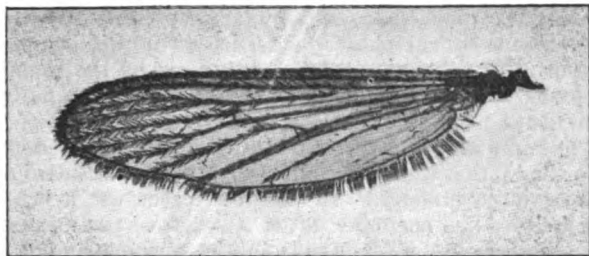


FIG. 3.

CULEX FATIGANS. WIED. (*Zwerflug. Insect*, p. 10.)

Like most of the earlier descriptions of the Culicidæ, Wiedemann's description is very incomplete, but it corresponds in all points given so absolutely with Major Ross' grey mosquito that there can be, I consider, no reasonable doubt of their identity. Ross' species has, however, I understand, been identified by some of the Italian entomologists as *C. pipiens* L. I therefore append to Wiedemann's description the points which completely distinguish the present from that species.

Wiedemann's description is as follows:—

"Fuscous; the thorax with two stripes; the abdomen white-banded; the legs yellowish. Length: 2 lines. *Habitat*—The East Indies. Antennæ brown; palpi and proboscis yellowish; brownish at the apex in certain lights; lower part of the face white. Thorax brown, with two very dark linear stripes; pleuræ yellowish. Every segment of the abdomen has a white band at its base. Wings limpid with brown scales, and with a fringe, whitish in certain lights, on its inner edge. The legs, in certain directions, appear whitish."

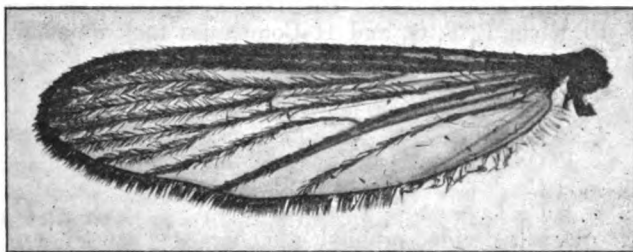


FIG. 4.—CULEX PAPIENS.

To show the differences of size and venation to *C. fatigans*. All four wings are photographed at the same magnification.

This species closely resembles *C. pipiens* L., but is much smaller, and may be further distinguished at a glance by the dark lines on the dorsum of the thorax, which are entirely wanting in *C. pipiens*, alike in fresh and rubbed specimens. In the denuded thorax, the difference is even plainer, the ground colour in this species being of the tint of old ivory, with a

median and two lateral brown lines, the latter being much the most conspicuous. In *C. pipiens*, on the other hand, the denuded thorax is black without markings. In addition, the lighter basal bands of the abdominal segments are much narrower, and not so yellow. Lastly, in the wing of *C. fatigans*, the second and third longitudinal veins have distinct scaly extensions running back into the first basal cell, beyond their points of origin from the first longitudinal and supernumerary transverse veins respectively, while these spurs are quite wanting in the wing of *C. pipiens*.

Although I have had several responses to my appeal for specimens of mosquitos in the Indian medical papers, only one package has as yet come to hand, viz., some kindly sent by Captain Victor Lindsay, I.M.S., containing two species from Bakloh. One of these was *C. fatigans*, which shows that the species extends to a considerable elevation in the hills.

His other species corresponds almost absolutely to *C. notoscriptus* described by Skuse from Australia. The only point of possible difference that I can discover is that whereas Skuse describes certain white bands on the joints of the tarsi as basal in position, these bands in Captain Lindsay's specimen involve slightly the apex of the contiguous joint. It is, however, obviously possible that this slight participation may have been overlooked by Skuse, although his description is very complete and minute. Should they prove identical, the geographical distribution is at least peculiar.

THE COMPARATIVE VALUE OF CERTAIN DRUGS IN THE TREATMENT OF MALARIAL FEVER.

By ANDREW DUNCAN, M.D., B.S.LOND., F.R.C.S.

Physician Branch Hospital Seaman's Hospital Society.

FOR the last three years of my service in India I have kept notes of the value of certain remedies in the treatment of malarial fever in India, the publication of which may be of some interest. The results in 367 cases are here accordingly detailed.

Drug	No. of Cases	Average Period during which it was taken for the Cessation of the Fever	Longest Period necessary for taking it for the Cessation of the Fever	Failure
Liq. arsenicalis ..	18	1.11 days	3 days	22.2 %
Bonduc nut. ..	11	1.66 "	8 "	18.1 "
Siounea ..	24	1.8 "	7 "	25 "
Asaprol ..	9	1.8 "	3 "	44.4 "
Quinine ..	78	2.11 "	8 "	2.05 "
Acorus ..	7	2.2 "	5 "	33.3 "
Nim bark ..	21	2.3 "	7 "	18 "
Choutha ..	8	2.4 "	6 "	25 "
Creasote (externally)	15	2.5 "	5 "	26.6 "
Berberis ..	25	2.6 "	7 "	50 "
Narcotine ..	66	2.77 "	7 "	1.06 "
Kreat ..	42	3.26 "	7 "	50 "
Phenocoll ..	17	3.4 "	7 "	18 "
Gulancha ..	6	3.83 "	5 "	33.3 "
Inderjao ..	20	4.6 "	8 "	5 "
Total ..	367			

These results, such as they are, are all derived from the effects of different drugs on Goorkhas, so that any idiosyncrasy due to race and locality may be eliminated. The number of cases cannot, of course, be held to be sufficient to draw decisive conclusions from, but leaving out those in which the particular remedy has only been administered in a small number of instances, let us consider the results in siounea, quinine, nim bark, berberis, narcotine, kreat and inderjao. With the exception of siounea; quinine gives the best results.

Siounea is a patent drug advertised by Messrs. Stannistreet & Co., chemists, of Calcutta, as being particularly efficacious, and made from a plant growing in malarious districts. I wrote to them and asked them the name of the plant, but was unable to obtain it. I then had the drug analysed, but the analyst concerned was unable to inform me of the essential ingredient. Though high up in the list of good results, yet I would point out that in 25 per cent. of the results failure occurred. It never succeeded once in checking the fever where it was at all severe. It is only of use in mild cases.

Quinine proved not only by the small number of its failures, but by the few number of days necessary to obtain a cessation of the fever, the best of the drugs where results were obtained from cases of twenty and upwards.

Nim bark, or the bark of the *Azadirachta indica*, was given either powdered in one drachm doses three times a day, or as two ounces of a decoction prepared by boiling two ounces of the bruised inner layer of the bark in a pint and a half of water for a quarter of an hour. It was of the drugs of which the larger number of records was obtained, the most successful after quinine as regards duration of treatment, but had five times as many failures.

Next in success came berberis.

Narcotine, recommended by Sir William Roberts, was tried in 66 cases in doses of gr. 2-3, was less successful as regards the length of treatment than quinine in the proportion of 2·77 days to 2·11, but the failures were only 1·06 per cent. as distinguished from 2·05 per cent.

Kreat, or a tincture prepared from the *Kreat halviva*, has been highly recommended by Bombay physicians. It, however, was necessary to give it for a longer period of time than any of the preceding, whilst in half the number of cases in which it was employed it failed to produce any effect.

Inderjao (*Wrightia anti-dysenterica*) was alluded to at the First Indian Medical Congress at Calcutta, December, 1894, by Dr. L. P. Gomez, Bombay, for "its marvellous effects as a prophylactic in malaria." I did not try it as a preventive, but as a curative agent in twenty cases; it was less than half as effectual as quinine, whilst the patients disliked it for its nauseous taste.

The other drugs employed were not used in sufficient numbers to draw any definite conclusions from. One remedy, however, was successful in the stoppage of the fever, viz., the rubbing in of creasote into the axilla. Fifteen drops were rubbed in for twenty minutes every day. A few minutes elapsed, and then a profuse perspiration broke out. In 26·6 per cent.

of the cases in which it was employed, it failed to stop the attack.

Chouthea is a mixture of quinine and sulphate of soda. It is very nauseous and not very successful, notwithstanding its laudatory advertisement.

My plan was to give each drug for seven to eight days. If at the end of that period no effect was produced, other means were then employed. In severe cases, the most successful by far was the exhibition of quinine by enemata, in addition to quinine by the mouth; this rarely failed. Five grains by the mouth were given night and morning, and twenty grains by enema at noon.

Quinine and carbolic acid has been strongly recommended; this was tried, but was not so successful as the quinine enemata. In eighteen cases the average case by the enemata after a week's preliminary quinine was in 5·14 days. In the quinine and carbolic acid treatment, after treatment by some other drug for a week, the cure was effected in 8·13 days.

In some cases the fever persisted in spite of all remedies, and the only method successful in stopping it was sick leave to another district.

With regard to the prophylactic treatment of malaria, the results I obtained from the following drugs were as follows. The drugs I used were quinine, quinetan, cinchona febrifuge, cinchonidine, arsenic, atees, narcotine.

With regard to arsenic, no benefits were obtained from it. In 1886 four companies of the 23rd Pioneers took from 5-10 minims of liq. arsenicalis during the months most liable to malaria; 28 cases occurred in these companies, 26 cases occurred in the remaining four companies. In 1887 the right wing of the 14th Sikhs took 6 minims daily from September 20 to November 16; the left wing none. The right wing had 8 cases, the left 9. In 1889 two companies of the 14th Sikhs were again placed on a ration of arsenic; the remaining companies not taking it were rather freer from malaria.

Quinine and cinchona febrifuge. Here in one year A and B Companies of the 14th Sikhs from August 2 to October 31st took respectively 3 grains, increased in the last week to 5 grains, of quinine and cinchona febrifuge respectively. Up to the week ending October 10, when E, F, G, and H Companies took quinetan, the results were as follows:—

A Co. taking quinine	10 cases malarial fever
B Co. " cinc. febr. 11	" "
E Co. }	{ 24 " "
F Co. }	{ 18 " "
G Co. }	{ 21 " "
H Co. }	{ 13 " "

Here a decided prophylactic effect was caused.

In 1896 50 men of the 2nd P.W.O. Goorkha Rifles took 3 grains of quinine daily, and 50 men 2 grains of narcotine.

The men taking quinine had not a case of fever.

The men taking narcotine had malaria in 3 per cent. of strength.

The men taking no drug had malaria in 6·5 per cent.

In 1897—

50 men taking 3 grains of quinine had malarial fever nil.

50 men taking 2 grains of narcotine had malarial fever in 6 per cent.

Remainder of regiment taking no drug had malarial fever in 9.8 per cent.

As regards the other drugs, I have no results of any value to offer.

Conclusion.—In the curative treatment of malarial fever no drug has yet been found to supersede quinine. It is especially valuable in severe cases when administered by enema. In the preventive treatment, as far as India is concerned, quinine again holds the field.

MICROSCOPIC EXAMINATION OF DYSENTERIC STOOLS.

By A. B. DALGETTY, M.B., C.M.
South Sylhet, India.

THE pathology of dysentery and its allies is of great interest, but up to the present time it is rather obscure. Hence any light that may be thrown on the subject must always be welcome. We want facts, not theories or statistics, and it is with the hope that the following facts may be of service to other workers in the same field that I venture to publish this short note.

As the interest lies in the microscopic findings, only a bare outline of the case need be given.

The patient, a young tea-planter, had been suffering for ten days from a dysenteric discharge which he attributed to two causes, namely, a wetting got while out at work, and following closely on this, a chill caught while lying uncovered in bed in the draught of a window.

After several days of diarrhoea, tenesmus and pains in the abdomen, blood began to come with the motions. He took to bed for two days, was treated by the native doctor, and improved so much that he returned to work: but the looseness of the bowels started afresh and then he came under my care.

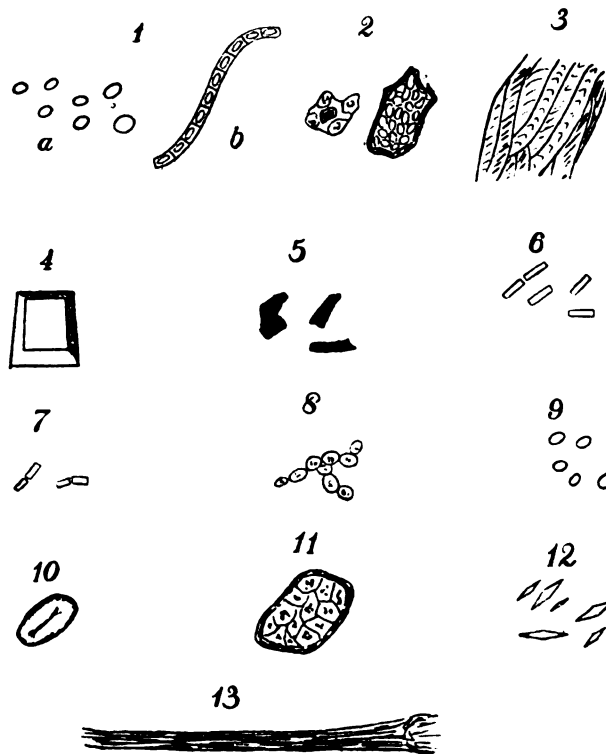
It is unnecessary to give details of the patient's general condition at this time; it will suffice to say that all the symptoms were those of dysentery with this important exception, that there was no blood in the stools.

Moreover, I gathered from his account that at no time of the illness had there been the typical dysenteric discharge of bad-smelling, scanty, gelatinous, blood-streaked mucus. The stool was more abundant than that of ordinary dysentery, and contained watery mucus and *débris* but no blood.

I regarded the case rather as one of colitis than of true dysentery, although there was no rise of temperature as one should have expected to find in colitis, and although at an earlier period blood had appeared in the motions and the other symptoms were those of dysentery.

After two 40-grain doses of pulv. ipecacuanhæ, given on two successive days, the discharge became less offensive and showed of a yellow colour, indicating the presence of fresh bile, while the desire to go to stool and the tenesmus greatly diminished. Following this, pil. plumb. cum opio (B.P.), gr. v. each, was given for three days—three pills first day, two pills second day, and one pill third day. No more treatment was required and recovery was uninterrupted. Milk and milk puddings was the diet throughout the treatment.

Below is a drawing of the microscopic appearances found in the stools.



X400

Description: (1) *Bacillus subtilis*, (a) spores, (b) jointed thread, each segment containing a spore. (2) Fatty-looking cells. (3) Vegetable tissue. (4) Crystal of ammon.-magnes. phosphate. (5) Jet-black particles. (6) *Bacterium coli commune*. (7) Rapidly-moving (vibratory) bacteria. (8) Yeast cells. (9) Rapidly-moving cocci. (10) Starch-grain. (11) Salivary gland-like cell. (12) (Leyden's) Crystals. (13) Hair.

Besides these substances there were irregular masses of epithelium, *débris* and mucus scattered over the field and gathered into heaps of a canary-yellow colour. No amœbæ were discovered.

Fig. (1) represents what I take to be *bacillus subtilis*. Fig. (5) shows perhaps minute particles of carbon. Fig. (7) depicts a form of bacterium which was fairly abundant, and which moved about with great rapidity, presumably by means of cilia, although I was unable to see these. Fig. (9) shows cocci, also numerous, and also in rapid motion. Fig. (12): it is chiefly with the object of calling attention to these crystals, if they may be so called, that this note was written. They were very abundant over the whole microscopic field, quite colourless, and varying in size from the smallest visible point upwards. They could hardly be described as crystalline in structure, rather mucine-like, but every one of them preserved the same finely pointed octahedral form.

They appear to correspond exactly with the crystals found by Leyden in cases of asthma,¹ and hence also

¹ "Hamilton's Pathology," vol. ii., part 1, pp. 66-67.

with those described by Friedreich, and by Charcot and Robin.¹

These crystals are not figured in the drawings of normal fæces, and there was nothing in the diet of the patient or in the drugs taken which would account for their presence. One dose of salol had been given, but salol crystallises in large irregular squares or oblongs, while carbolic acid has long needle-shaped crystals, and those of salicylic acid are rod-shaped.

Whether these crystal-like bodies had any causal relation to the dysenteric condition it is impossible to say; there is some analogy between a bronchitis and a colitis, both of which are caused by some kind of irritation, but I do not press the point, as Leyden's theory of these crystals as the active agents in asthma and bronchitis is not generally accepted.

I only state the fact as I found it in the hope that others may be able to throw some light on the matter.

It seems to me that we are much in want of drawings of things found in tropical diseases, although we may not be always able to interpret their significance. We are far from reference-books of all kinds, and indeed the store of those on tropical medicine is yet only of the most meagre description.

BACILLUS OF LEPROSY IN THE BLOOD.

By EDWARD G. HORDER, F.R.C.S. EDIN.

WITH plenty of patience and perseverance the bacilli of leprosy in the blood can be demonstrated. It is well known, lepers suffering from either the anæsthetic or tuberculous variety, or both combined, have at varying intervals sudden exacerbations of the disease, usually accompanied with a good deal of inflammation, and a smart rise of temperature.

When it is desired to demonstrate the bacilli in the blood, it will be found this is the best period to make the attempt, and the punctures should be made at the inflamed edge of an anæsthetic patch, or into a newly inflamed nodule.

I strongly advise taking at least twenty or thirty spreads from each patient, and the use of cover-glasses in preference to slides. After pricking the skin, touch the exuding drop of blood with a glass and cover a second; when the blood is properly spread, separate cover-glasses evenly and carefully, and proceed in the same way until you have at least twenty well-spread glasses.

After trying various stains, I am inclined to think the carbol-fuchsin is the best, and that staining the specimens in the cold solution for twenty-four hours gives infinitely better results than heating for a few minutes over a spirit lamp. Therefore, so soon as the blood is dry on the cover glasses, place solution in a glass dish—as used for developing photographic plates is good—and place the covers face down on its surface and protect with a sheet of glass. If this is done, say, early in the morning, they are ready the next for the second step in the process, viz., wash, pass through 25 per cent. H₂SO₄, wash again, dry

and counter stain with methylene blue. Mount in xylol balsam. Examine with $\frac{1}{4}$ objective and follow up with $\frac{1}{2}$ oil immersion.

The bacilli are usually found in the leucocytes; now and again they may be seen in the plasma.

I have been successful in obtaining one good specimen out of four spreads, but I advise taking a larger number, which needs but little longer time, and running through them all with the $\frac{1}{4}$ objective (and No. 4 or 5 eye-piece) before using the oil immersion lens.

A FURTHER NOTE ON PRICKLY HEAT.

By T. FREDERICK PEARSE, F.R.C.S. ENG.

THE opinion that this disease is an affection of the sebaceous glands has been called in question. It is perhaps wrong to call it a seborrhœa if by that is meant an increased flow of the secretion of the sebaceous glands, but I think it is an acute distension of those glands by their own secretion. Prickly heat only occurs on those parts of the body where sebaceous glands exist. It will not be found on the palms of the hands or the soles of the feet—the borders of these surfaces being often distinctly outlined by the eruption. It does not appear to develop easily where there are large hairs, as on the scalp, armpits and pubes. It is often associated with distinct acne-like spots. Just as sudamina are the distended mouths of the sweat glands, so I consider the pin-point heads of prickly heat the irritated openings of the sebaceous glands. Sudamina are occasionally seen associated with prickly heat because of the profuse sweating also present, but are much more common in rheumatic fever and as the result of severe training for athletic exercises. Why prickly heat is not common on the scalp appears to be explained by the open channels alongside the large hairs giving vent to the secretion of the sebaceous follicles. Distension of large sebaceous follicles with subsequent inflammation is acne.

In ordinary seborrhœa there is an increase of sebaceous matter, and the more the secretion is removed by soap or its equivalent the more the activity of the glands is, to my mind, stimulated. Many analogies could be given to support the principle that the removal of the secretion from an organ tends to make that organ pour forth more products, or perhaps it would be more correct to say that the presence of the secretion of an organ inhibits in some way the further formation of its material. My explanation of prickly heat was therefore an acute distension of the small sebaceous glands connected with the fine downy hairs covering the greater part of the surface of the body, and the presentation of their blocked-up ducts as minute, more or less conical, reddish heads on the surface of the skin. It may be that the cells of the horny layer imbibe water and swell so as to occlude the orifices. It is not correct, however, as Dr. Gray says, that "where the perspiration evaporates rapidly or can be drained away by absorbent underwear there is little or no prickly heat." The complaint is common enough on the hands, and in bad cases frequently on the face. It is very much less marked on the legs and feet. Whatever clothing be worn the disease seems

¹ *Ibid.*

to have a peculiar predilection for the forearms, especially on the radial side.

With reference to treatment, there appears some misapprehension. I think that the skin requires lubricating, and the best material for this purpose is undoubtedly lanoline. Personally I have found the mixture with it of almond oil and menthol give good results, but it is quite a mistake to suppose that so much need be put on as to spoil all one's clothes. As a matter of fact, the mixture massaged into the skin nearly all disappears in the course of a few minutes, and the ridicule of the negro habit cast by Dr. Gray shows he has had little experience of these materials. Undoubtedly removal to a cooler climate is the best remedy, but to those who cannot afford this or the former treatment, I would suggest a solution of perchloride of mercury and menthol, or hyposulphite of soda solution with menthol. The laundry terror conjured up by Dr. Gray has no real basis if the mixture of lanoline, almond oil and menthol be used as I have directed. It need not be used extravagantly, but to avoid the soiling of clothes it must be massaged into the skin. The distress arising from the disease is, in my experience, sometimes so acute that patients will be willing to try anything, and those who have once experienced the benefits of the above anodyne lubricant do not hesitate to repeat the use of it. It must not be imagined that it cures—leave off the treatment and the disease will return in a day or two—but it is the means of affording the very greatest relief.

MULTIPLE SCLEROSIS FOLLOWING AN ATTACK OF MALARIAL FEVER.

By E. A. CHARTRES, L.R.C.S., R.C.P.I.

Medical Officer North Cachar, District Tea Gardens, N.E. India.

GASETTY, a coolie, aged 32 years, working on one of the gardens of my district, got an attack of fever with vomiting and diarrhoea, which was treated by quinine and astringent mixtures by the native doctor; his temperature had varied between 99° and 103° from the date of his attack, August 15, 1898, until the 20th. On this fifth day of illness he complained of giddiness and headache, flashes of light before his eyes, and stabbing pains in his limbs, with great weakness. I saw him on my rounds for the first time on the 23rd; he was quite paralysed in his lower extremities, arms not affected. There were well-marked nystagmus, oscillation of the head and hand tremors, which were quite violent when any movement was attempted. He spoke in a very slow, deliberate manner when asked a question. (This way of speaking, his friends said, was unusual.) The spleen was enlarged about an inch below ribs, other internal organs normal; pulse small volume, about 70 to the minute. Patient complained that he slept badly owing to the pains he had in his legs. He was ordered a bromide mixture, which was discontinued when his sleep improved, and a mixture of quinine, nux vomica and arsenic substituted. He had little or no fever after this, never going above 100°. From the beginning of September he began steadily to improve, and was able after six weeks to move about a little with assistance. At the

present time he is doing the full task of an able-bodied man and seems in perfect health. The only remaining symptom of his illness is, if he is suddenly struck on the shoulder, some slight nystagmus is observed and oscillation of the head.

DANGERS TO HEALTH ON BOARD OF PASSENGER STEAMERS.

By D. E. ANDERSON, M.D. PARIS, M.B., B.A., B.Sc. LONDON.

THE following observations, from personal experience, taken during four voyages on board of eight steamers belonging to three of the largest Companies whose ships go to all parts of the world, may be of use, not only to intending passengers, but to the Companies themselves, if the dangers herein exposed be avoided by the former and removed by the latter.

CLASSIFICATION.

These dangers may be classified under the following headings:—

Class A.—Contagion from—

- (1) Tubercular fellow passengers, or those affected with dermatitis.
 - (2) Want of proper hospitalisation of sick passengers and sailors.
 - (3) Inadequate accommodation for troops invalided home.
 - (4) Water-closet leakage.
 - (5) Cattle-pens and menageries.
 - (6) Water-closets on the lower decks.
 - (7) Desiccated sputum on deck.
 - (8) Impure water taken on board at ports-of-call.
 - (9) Food and drinks partaken of ashore.
 - (10) Miasma in harbour.
 - (11) Mosquito bites.
- Class B.—Danger from imperfect ventilation.
Class C.—Danger from unsuitable cabins.
Class D.—Danger from the heat, and from exposure to the sun's and the moon's rays, in the tropics and in the torrid zone.
Class E.—Danger from want of exercise.
Class F.—Danger from uncontrolled medication.
Class G.—Danger at the hands of inexperienced medical officers.

CLASS A.

- (1) *Contagion from tubercular fellow-passengers and those affected with dermatitis.*

It is now admitted that tubercular disease of the lungs may be caught by sleeping in the same room with a tubercular patient, especially if that room be small and badly ventilated, or one's own lungs be weak. That kind of fellow cabin passenger is to be met with more frequently on certain voyages than on others, and as long as isolated cabins on deck are not provided for him and others affected with contagious diseases, the steamship companies are to blame. It may be argued that few passengers would beforehand declare themselves tubercular, but on the slightest suspicion on the voyage, the ship's doctor should be empowered to examine the delinquent, and either compel him to pay for and occupy the whole cabin, or go to the one specially set apart for him.

The same danger exists in the cases of skin diseases. The towels and pillows get mixed, and eczema and other serious dermatitis are known to have been caught in this wise.

(2) *Contagion from want of proper hospitalisation of the sick.*

Few steamers have special accommodation for sick sailors and passengers. As a rule, if a passenger develops a contagious disease during the voyage he is transferred to a cabin as remote as possible from the saloon, and is there kept in quarantine until he can safely join the others again if the attack be mild; but if the disease be serious, he is landed at the next port-of-call, and sent to the quarantine hospital, and his cabin is disinfected and closed. The danger of complications, the expense and terror at being left behind at the mercy of strangers, with a foreign doctor in charge (as a rule, a minor star), render the situation all but maddening.

On board of some steamers going long distances, a cabin on deck is reserved as a hospital, but it is generally used for surgical cases. It is, to my knowledge, that on board of a large steamer of a much-patronised company the third-class women's general cabin was turned into a hospital, and the women were put into the second-class. Two patients occupied the room—what their ailment was I do not know, but it must have been serious for one died on the third or fourth day—and as the door of communication between it and the saloon, was partly constructed with fixed venetians, the cabin was ventilated through the saloon, and the latter received in return the foul air from the former. And yet some twenty or thirty passengers daily sat down to their meals in that saloon, and slept in the other cabins opening into it. How they escaped contagion is a mystery, or was it due to the sea air, that panacea with which companies and passengers tempt Providence?

(3) *Contagion from inadequate accommodation for troops invalided home.*

It is a very common occurrence to pick up at different ports of call batches of soldiers invalided home, some in the last stages of disease, whose only chance of getting well is the sea voyage, but which often proves their last (unaccomplished) journey in this life. It would be inhuman to refuse to embark them, and thus in time of war or soon after, as many as a hundred or more of these poor fellows are located on the lower deck, where cargo is generally put, and which becomes a vast hospital ward, with convalescents and dying breathing the pure sea-air brought to them by the ventilating shafts and windsails, but exhaling through the hatchway, into the second and third-class cabins on the deck above, their poisoned breaths. The stench which comes through these exits after a stormy night when the ventilators have perforce been closed, is strong enough to knock one down. In fact the whole forepart of the vessel where the hatchways open, becomes contaminated.

(4) *Contagion from water-closet leakage.*

As a rule the first and second class water-closets are on the lower deck, at a distance from the cabins—

although I know one steamer in which they open directly into the saloon—and those of the third-class and crew are on the upper deck, and sometimes just over the third-class cabins. In the latter case, under severe stress of weather, when the decks are apt to warp, the joints of the planks get loosened, with the consequence that a leakage from the water-closets takes place upon the passengers in the cabins underneath. This occurred on board of one of the largest liners from the East after a severe storm in the Indian Ocean, and for several days after, the berths situated below could not be used.

(5) *Contagion from cattle-pens and menageries situated on the upper deck.*

The same danger of percolations of fetid liquid through the fissures of the deck on to passengers beneath arises from the menageries which are invariably to be found on steamers from the East in that portion of the deck reserved to the third-class passengers. The cages of these quadrupeds and birds were cleansed once or twice a day, and at those times or in stormy weather the neighbourhood was unapproachable.

(6) *Contagion from water-closets situated on the lower deck.*

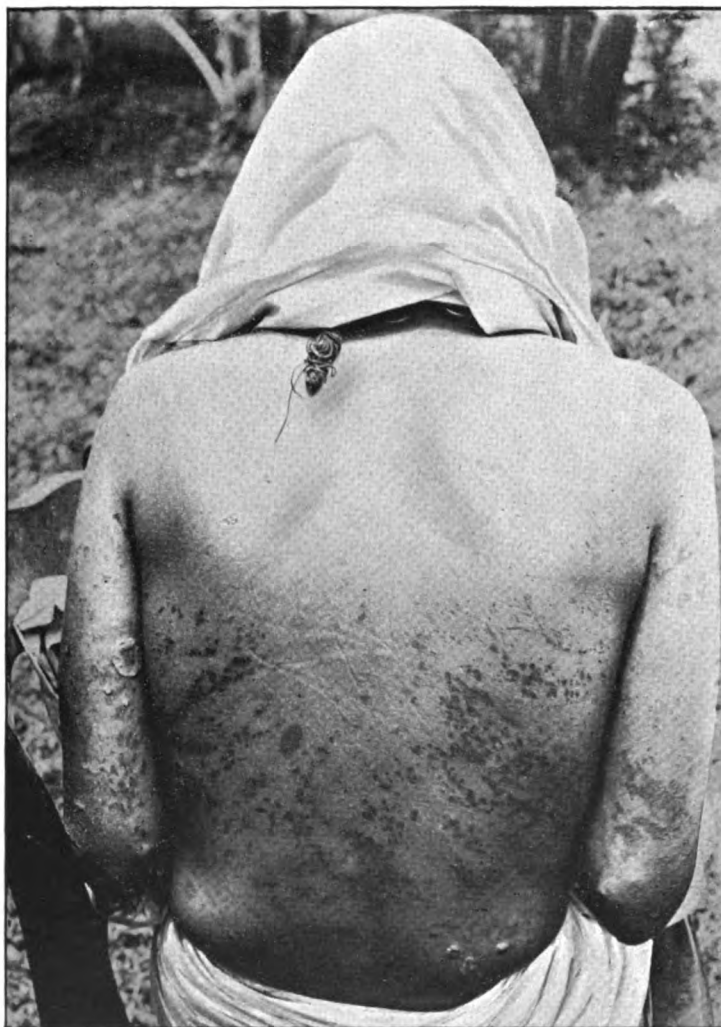
In stormy weather, or if for some reason or another the hulk of the vessel has been lowered into the water below a certain level, the outlet of the drains naturally gets below water-mark, and the storm-valves are apt to get dislocated or jammed, and a "refoulement" of noxious gas, or of the contents of the pan, is the consequence. It seems to me the naval architects should be able to solve that difficulty.

(7) *Contagion from desiccated sputum on deck.*

On most liners smoking on the promenade deck is prohibited, but this regulation is invariably broken, the captain and officers themselves sometimes setting the example. As spittoons are dispensed with, the deck very quickly becomes filthy, and in the tropics and torrid zone the desiccated sputum becomes a source of danger. I must own that this dirty habit does not prevail on board of English and American steamers.

(8) *Contagion through drinking impure water taken on board at ports of call.*

This is a serious source of danger, and the wonder is that not more harm is done by it. Anyhow most cases of typhoid, dysentery, filaria, and cholera on board emigrant vessels could be traced to it. I know for a fact that the ships in the harbour of Port Louis, Mauritius, take their water from a fountain which, until a dozen years ago, derived its supply from the Grand River dam—and this dam was afterwards condemned on account of the impurity of its water, and the fountain obtained a new supply from another dam some three miles up the river; but until this very day the coolies bathe a mile farther up the stream, and dhobies wash in it the clothes of the dead and the dying. I saw with my own eyes in 1889 a score of these dhobies washing clothes in a rivulet on the watershed of the island. Is it to be wondered at that malarial and typho-malarial fevers have become endemic in Mauritius? Surely the laws against

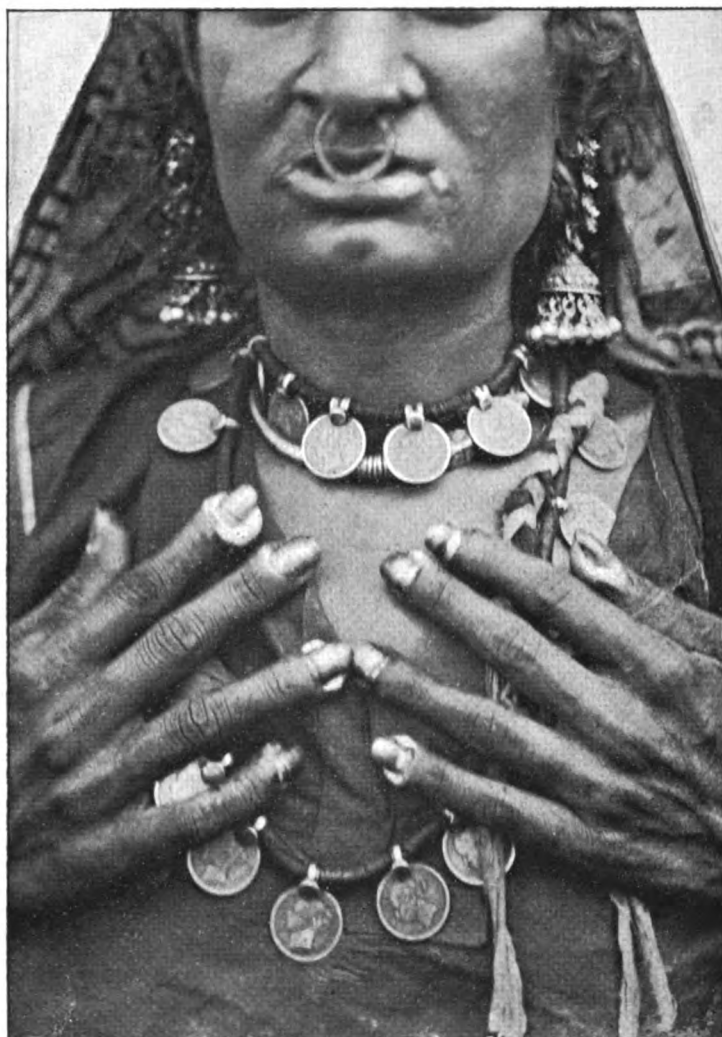


YAWS.

Photograph of a Hindu woman, showing stains left by eruption when faded as well as some active granulomata, contracted in Fiji.

By Dr. H. N. JOYNT, Fiji.

For descriptive letterpress see plate in September issue.



YAWS.

Showing onychia and sores at angles of mouth and nose.

Photograph of a Hindu woman suffering from Yaws, contracted in Fiji.

By Dr. H. N. JOYNT, Fiji.

For descriptive letterpress see plate in September issue.

the pollution of potable waters should be sacredly enforced everywhere. True, nowadays, passengers drink mineral waters or water condensed on board, but what about emigrant vessels and troopships?

(9) *Contagion from food and drinks partaken of ashore.*

Travellers cannot be too strongly put on their guard against the food they eat ashore at ports of call. The curry-and-rice, the chutnies, pickled fish, mango salads and other Indian dishes, not to speak of the fresh and preserved local fruits, are a great temptation, and most Europeans whose stomachs are not accustomed to digest such highly spiced food or juicy fruit have to pay pretty severely for it during many days after. Another danger, to which I myself nearly succumbed at Port Elizabeth, is to be found in the canned food which hotel-keepers foist upon their visitors under the disguise of made-up dishes, when their dining-rooms are besieged by an unexpectedly large number of customers, as on the sudden arrival of three large steamers in the roadstead, whose passengers all wish to spend the few hours' stoppage ashore. Of course the last comers get bones, or they must be satisfied with tinned lobster, curry-and-rice. The painful gastritis that ensues during the next few days on board is not the fault of the ship's cook. I need not dilate upon the danger which lurks in the variety of cock-tails, iced drinks and other indigenous beverages with which the passenger as well as the sailor too oft quenches his thirst.

(10) *Contagion from miasma whilst in harbour.*

There are certain tideless harbours known to be very unhealthy, regular cesspools, some of them, exhaling disease in the radiating mist that rises under the influence of a torrid sun. Whilst the ship is coaling or taking cargo, the passenger must either sleep ashore or take his chance of resisting the baneful miasma, but very often it is more dangerous to sleep ashore than on board.

(11) *Contagion through mosquito bites.*

In the harbours just described, or those surrounded by dense vegetation, or situated at the mouths of rivers in the tropics, mosquitoes are plentiful during the hot and rainy months. Woe to the ships that then lie close to the shore, or are tied to the quays, the mosquitoes will do sad havoc (in spite of nets and curtains), amongst the passengers and crew. Certain species among these creatures have, according to recent researches, acquired a notoriously bad character, and are said to be the introducers of the filaria, hæmatobia, and malaria parasite, into the human body.

CLASS B.

Danger from imperfect ventilation.

In former days when steamers used to be constructed with cabins on each side of the saloon, and the sole means of ventilating the latter was through the skylight and the companion stairway, it often happened when these were closed that the atmosphere became close, and added to the passengers' discomfort. But few ships now are built on that plan.

From what has already been said concerning the poisoned atmosphere of the lower decks transformed into hospital wards, it is evident that the present system of ventilation is defective. The revolving fans lately introduced on board every large steamer have produced only a partial success; they certainly create a movement in the atmosphere, but no number of fans revolving in vacuo will cause a draught, and the lowest decks on board the great Leviathans which have, within the last ten years, been launched, are virtually exhausted air-chambers under certain conditions, as in stormy weather, when the ventilators are either blocked, removed, or blown away, or when the ship is in harbour, or in a dead calm.

Even with a fair breeze blowing down the shafts, no reviving draught is felt from a fan fixed in some distant corner, far away from the ventilator, and at the end of an intricate maze of corridors. Surely steam power could be employed, as on board of turret ships, to force fresh air into the lower decks, and to aspirate thence the impure atmosphere.

CLASS C.

Danger from unsuitable cabins in cargo boats taking passengers.

Well and good if the latter take passage on board such boats with their eyes open; they must not afterwards complain if the voyage is made in great discomfort, amidst nauseating smells and in small cabins, some of which are actually situated over the propellers, the incessant rotations of which act as a drill on one's body, causing every nerve and fibre to vibrate as in a tetanic spasm.

But most people are not aware that the Company's object is to carry as much cargo as possible, devoting all available space to it, and leaving the passenger's comfort out of consideration. At some distant seaport he is transferred on board a smaller steamer, and his miseries are increased tenfold. Woe to him if he falls ill; he cannot lie down in his cabin on account of the wretched screw, nor can he walk about during his convalescence, for there is no deck. Such boats should not carry passengers at all.

CLASS D.

Danger from the heat and from exposure to the sun's and the moon's rays in the tropics and in the torrid zone.

As we should expect in certain parts of the globe and at certain seasons, the heat even at sea is intolerable. In the Red Sea, for instance, the months of August and September are dangerous, and both there and under the equator, sunstroke, gastric, intestinal and hepatic disorders are of common occurrence. Abscess of the liver, too, is not infrequently met with in those who are obliged to expose themselves constantly during the year, like the engineers on board steamers passing through the Red Sea, to the vicissitudes of cold and heat in addition to a prolonged sojourn in the engine room.

That rarest of occurrences, the moonstroke, is as a rule only to be met with in the subtropical regions, when the bright moonlight on a warm night tempts

the passenger or sailor to sleep on deck exposed to its rays.

The *Lichen tropicus* is another penalty the novice passenger has to pay who takes a tepid sea-bath in the torrid zone.

CLASS E.

Danger from want of exercise.

Most men take plenty of exercise by walking up and down the decks and engaging in games, but some ladies, either through sickness or disinclination, do not imitate the men, and they are apt before the voyage is over to suffer from jaundice or some other hepatic and intestinal trouble.

CLASS F.

Danger from uncontrolled medication.

Patented medicines and other mixtures, and lozenges containing one or more of the following ingredients: chloral, bromides, opiates, cocaine, chloroform, hydrochloric acid, are very much in vogue among some travellers who suffer from sea-sickness, and when partaken of too freely are apt to produce serious effects.

CLASS G.

Danger at the hands of inexperienced medical officers.

Some of the English liners employ only fully qualified medical men who have had a fair amount of practical experience, but others are satisfied with L.S.A.'s, or *officiers de santé*, with but a limited knowledge of accouchement and surgery, with the consequence that when a serious case presents itself the doctor is nonplussed, and the patient runs the risk of dying or of remaining a cripple for life.

It is deplorable that such a state of things should be permitted. The Medical Council should prevent any but doubly qualified men from serving on board any ship.

I have come to the end of my notes, and I have no doubt other dangers exist which must have come under the notice of other passengers, and I dare say the ship's medical officer could tell us of others still which can only have been observed by him.

Paris, Oct. 2, 1899.

ON TROPICAL ANÆMIA, AND ITS RELATIONS TO THE LATENT AND TO THE MANIFEST FORMS OF MALARIAL INFECTION.¹

By ALBERT PLEHN, M.D.

Physician of the Imperial Government in Cameroon.

It has long been a well known fact to the medical practitioner residing in unhealthy districts in the Tropics that a number of his patients, even at times when strictly speaking free from symptoms of disease, appeared however to be suffering, more or less, from poverty of the blood. One usually ascribed such a state to the effects of the tropical climate, or also to the so-called "process of acclimatisation," without seeking for an explanation as to the cause of this condition.

USUAL IDEA OF TROPICAL ANÆMIA UNTENABLE.

It has been proved through careful and methodical examination of the blood as to the amount of hæmoglobin present, number of corpuscles and specific weight (as lately carried out by Glogner, Van der Scheer, Hammerschlag, Eijkmann, and by Friedrich Plehn, in the case of apparently healthy inhabitants of salubrious districts in Java) that there is no such thing as tropical anæmia as described by the early observers. This is especially true on comparing the results obtained from an examination of newly arrived and of native individuals of different races.

AVOIDING SUNLIGHT CAUSES ANÆMIA.

The remarkable paleness of the inhabitants of European origin in the tropics is perhaps mainly to be attributed to the care with which the action of sunlight is avoided in tropical regions. The experience I gathered myself on a journey in the Dutch Indies twelve years ago agreed perfectly with these facts.

ANÆMIA NOT MALARIAL.

I was therefore all the more surprised, during the period of my first service in Cameroon, to observe a form of anæmia most clearly characterised not only through a reduction in the number of red blood corpuscles, but also by a diminution of the hæmoglobin percentage, and which cannot be accounted for as the result of malaria in the sense of Bignami and Manson. Bignami found that the pathological changes of anæmia were not present in the fœtus nor in the new-born of a mother suffering from malarial anæmia, and thereby supports his theory that toxic substances can hardly be the cause of anæmia, since these would pass with the placental circulation. He seems to attribute it to the direct action of parasites which do not get to the fœtus. Manson, on the other hand, ascribes the action to toxic substances supposed to be formed through the decomposition-products of the dead plasmodia. In the year 1896, at the 68th meeting of the "Naturforscher" in Frankfort, I was able to communicate the fact that the diminution of red corpuscles, and especially of hæmoglobin in the blood, occurs soon after the arrival in Cameroon, often long before the first attack of fever, and also that the negroes of the coast, who very rarely suffer from fever, are likewise affected with anæmia. I was unable at the time to offer any explanation.

OPPORTUNITIES OF OBSERVATION.

On again resuming my work in Cameroon, I continued to thoroughly investigate this question, which is of vital importance in judging of the possibility of acclimatisation and also of the fitness for tropical service in fever-struck districts. Through practical experience I have come to consider the estimation of the hæmoglobin as the best standard for the valuation of blood, and I determined the amount by means of Flaisch's hæmoglobinometer in the manner I have already described on a former occasion.

I was at the time unaware of the latest improvements in the apparatus, however, after a daily use of the same instrument, under the same conditions, and considering that the determination was always made twice, I do not believe that the error amounted often to two per cent.

¹ Paper read before the Berlin Medical Society on May 31, 1899.

Moreover, in many cases the blood corpuscles were counted, and special weight was laid on the specific gravity determination of the total blood. I shall return further on to the clinical importance attached to a comparison of the different results of these determinations. Owing to the great individual variations in the composition of the blood even at home, I determined the amount of hæmoglobin as soon as possible after the arrival of new Europeans—in any case within the first week, mostly within the first two or four days. The examination was repeated thereafter continually every four weeks, and even oftener if special conditions made it desirable. That I was able to examine and to control during two years, excepting in a few single instances, all the Government officials, I owe partly to the intelligence and interest shown by most of the latter, but especially to the untiring assistance I received also on this occasion from our Governor, v. Puttkamer, and from his substitute, Regierungsrath, Dr. Seitz. I was thus able to carry out continuous and accurate observations in the case of about fifty Europeans. The value of these observations, few as far as the number itself goes, increases considerably because it contains almost *all* the officials and not a number chosen from a larger material, in which case the selection must be always arbitrary or guided by chance.

Through the kindness of the commanders and of the doctors of our men-of-war outside, I was also able to examine a considerable number of officers and of men of the Imperial Navy, who, owing to the fact of their being on board ship in a river three km. wide, live under quite different conditions to the Europeans on land. Finally I examined natives of Cameroon, from other parts of the African West Coast, and from mountain districts without a doubt free from fever. All in all, I carried out during the last two years in Africa over 1000 double hæmoglobin determinations, about 120 specific gravity determinations, about 600 microscopical examinations of the blood, and determined in some 50 cases the number of blood-cells present. Hence it will be seen that what follows is the result of systematic work, and not arbitrary conclusions from superficial observations.

LOSS OF HÆMOGLOBIN WITHOUT FEVER.

The amount of hæmoglobin turned out in some of the cases, on first examining the blood within the first few days, to be rather small, inasmuch as the newcomer had already touched the West African Coast before his arrival in Cameroon. After four weeks the hæmoglobin diminished, often considerably, and then increased again during the following pause between two observations, sometimes to a point still higher than had been found at the first examination. In other cases this primary oscillation was only noticeable during the course of the first few months. The differences amounted to 20 per cent. at times, and this, it must be well understood, *before any trace of fever had made its appearance*. Only in extreme cases did I hear complaints of a slight feeling of heaviness in the head, lack of appetite, pain in the loins, or that the legs felt heavy. In one case the temperature, as measured in the armpit, rose to 37.6° and 37.7° C. in the evening. Organic changes, espe-

cially any swelling of the spleen, could not be detected. Several times I observed at this stage a relative increase in the number of white blood corpuscles, and more rarely the presence of a few nucleated red ones. Usually, as the process advances, one observes that during the weeks and months following the above-mentioned increase, the amount of hæmoglobin as a rule diminishes again to a greater or less extent, or fluctuates irregularly, or even remains stationary until, sooner or later, the first attack of malaria fever follows. The effect of each successive fever attack on the degree of the blood impoverishment is quite variable. On comparing the number of plasmodia with the extent to which blood-cells have been destroyed, one is struck by the inadequacy of Laveran's explanation, when he says that no more plausible reason (excepting direct loss) can account for the poverty of the blood than malaria, whose germs attack directly the blood cells. The presence, on an average, of two or three parasites in every field of view would justify one in Cameroon in qualifying the blood as already thickly colonised. Working according to the method I employed, one finds about 100 to 300 blood corpuscles in every field of view, if the slide has been successfully prepared, and using an oil-immersion ($\frac{1}{2}$). This would yield about 1 to 2 per cent. of infected blood-cells. The number of parasites is usually already considerably reduced on the day following the first strong doses of quinine. Hardly any isolated parasites are to be found on the day following the second dose, and the fever has stopped. Accordingly, one would expect a total reduction of only 2 to 3 per cent. in the number of blood disks, and therewith in the amount of hæmoglobin, even in the case of a serious infection. One finds, however, even when a much smaller number of parasites are present, perhaps a reduction of 10 to 15 per cent. hæmoglobin following immediately after the two days of fever.

In other cases, clinically quite as severe, it is quite impossible to detect any hæmoglobin reduction; nay, sometimes a slight *increase* of the blood pigment may be observed after the attack, and this quite clearly. This increase is due, without doubt, to the excessive regeneration of the blood caused by the reaction. Eight or ten days afterwards, during a perfectly normal convalescence, a diminution of 10 or 12 per cent. may perhaps again follow.

In short, *the anaemia observed in a particular case is often quite out of proportion to the severity of the single malaria attacks*, and, moreover, it is often out of proportion to the number of attacks within a short period.

In an earlier paper I communicated a case in which the amount of hæmoglobin fell to 36.5 per cent., although only two quite abortive fever oscillations had occurred in the previous months. Since then I have observed a similar occurrence. In the case of a nurse, as an instance, the hæmoglobin at the end of the first year of work during which she had only had a few times a slight fever, had sunk from 70 per cent. to 28.5 per cent. within two months, although the temperature of the body did not reach 38° C.

On the other hand, in some cases very severe attacks with numerous parasites would follow within pauses

of hardly twelve days, without any noticeable change in the constitution of the blood, naturally under proper treatment of the single fever attacks.

The above observations demanded some other explanation for the origin of the anæmia. *Ankylostoma* was out of the question as far as the Europeans were concerned, as they only drink filtered and boiled rain-water. Besides, all the local symptoms of "ankylostomiasis" were absent. This also applies to the coloured races, who, according to the investigations carried out here in Berlin by Zinn and Jacoby, often harbour indeed a small number of these intestinal parasites, without, however, showing any symptoms on that account. My brother has not found the parasite in Cameroon, and it must be in any case very rare.

In order to push the matter on, my first endeavour was to determine in what way the constitution of the blood is modified by those diseases which do not especially affect its elements.

RELATION OF HÆMOGLOBIN AND SPECIFIC WEIGHT OF THE BLOOD.

I had already previously tried to establish the ratio existing in general between the amount of hæmoglobin and the specific weight, in order to use it as a control in determining the former, thinking I might perhaps obtain a quotient useful for comparisons. As a result, I observed that with a percentage of 90 to 95 of hæmoglobin (as determined on an average in the case of a newcomer with my apparatus), the specific weight of the blood amounted to 1060-1063, the determination mixture being at a temperature of about 25° C.

Moreover, I found that a reduction of x per cent. hæmoglobin corresponds approximately to a fall of $\frac{1}{3}$ thousandths in the specific weight, and this as far as the loss could be traced back *only* to the anæmia we are now studying, or to the action of ascertained malaria attacks *alone*.¹

Naturally these comparisons cannot claim scientific accuracy; but, as you will soon see, the results are *clinically* especially valuable, when the normal quotient for a particular individual has been previously determined. Although the quotient in general, in Cameroon, expresses the above-mentioned ratio, the specific weight is unduly reduced as compared with the amount of hæmoglobin in cases of typhoid and dysentery, which are relatively free from complications. The amount of hæmoglobin remains then tolerably unchanged, a fact which proves that in this case the impoverishment of the blood proceeds at the expense of other elements, perhaps of the plasma. Even if this symptom be not *always* quite evident, it must be remembered that in Cameroon it is impossible to exclude the causes which affect especially the red blood corpuscles, in the presence of the other complaint. In the case of dysentery the modification of the quotient is usually so considerable that it may be most successfully taken into account in the differential

diagnosis, viz., if, in the case of a cachectic individual with diarrhoea, the specific weight of the blood is strikingly reduced as compared with the amount of hæmoglobin, one may be sure that *malaria* is not the direct cause of the patient's condition. This is important therapeutically, for the microscopic examination of the blood in such cases is often misleading.

On the other hand, excessive specific weight of the blood indicates in the same a decomposition of red cells, which is too extensive to be immediately counterbalanced through the natural process of elimination, and further, which cannot be immediately detected to its full extent (through counting of blood cells, and through hæmoglobin determinations) owing to the active blood regeneration which the reaction of the process instantly calls forth.

If, for instance, a newcomer's blood which on his arrival showed 90 per cent. hæmoglobin and a specific weight of 1063 at 25° C. goes down to 70 per cent. (that is, is lowered by 20 per cent.) after the settler has exposed himself for some time to the influence of malarial districts, one would expect a specific weight of about 1053. If this, however, only amounts to 1048 one will look for other diseases, especially in the organs of digestion. If, on the contrary, it amounts perhaps to 1057, one may well suppose that an active decomposition of the blood cells is going on, and it is then necessary to couple the examination microscopically, by counting the blood corpuscles, &c.

This process is naturally most noticeable in the cases of *blackwater fever*. I shall speak more extensively of this later on. Just now I wish to reiterate how efficiently we can be assisted in judging the character and the course of tropical complaints, through a thorough study of the physical composition of the blood. A former reference to this point seems to have met with little recognition.

EFFECT OF MEDICINES AND ELEVATION.

As for the treatment of anæmia, iron proved quite inadequate. Hommel's hæmatogen was often a little more efficient. Arsenic improved sometimes the appetite, and proved useful in this way, although it had no other advantage. In many cases the regular use of quinine ($\frac{1}{2}$ gram every five days) proved very successful; and in most cases a change of air to the *plains* turned out beneficial, even when the change was apparently no improvement as to neighbourhood. A sudden transition to the hill-stations is inadvisable in the case of highly anæmic inhabitants of the tropics.

In the very few cases in which I was able to study the action on the constitution of the blood of a stay in Buea (a Government station on the Cameroon range, situated at 1,000 meters) no increase in the amount of hæmoglobin was to be detected, as has been observed by others in European mountain stations, although Buea is certainly free from malaria and the nursing there is excellent. On the contrary, immediately after the return to the plains the amount of hæmoglobin had decidedly *diminished*, and this reduction only disappeared in the next few weeks. The amount of hæmoglobin in the blood of healthy coloured and European inhabitants of Buea whom I examined was the same as under similar conditions in the plains.

(To be continued.)

¹ On my return, I learnt with great interest that Diaballa had determined in the meantime the ratio between the specific weight of blood and the amount of hæmoglobin present, and had got a quite similar result. He found (according to Ehrlich) that 10 per cent. hæmoglobin corresponds to 4.46 per 1000 in the specific weight.

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THE

Journal of Tropical Medicine

OCTOBER, 1899.

THE OPENING OF THE LONDON SCHOOL OF TROPICAL MEDICINE.

THE London School of Tropical Medicine began its Session with an introductory lecture, delivered by Dr. Manson, which is published in this issue of the Journal. There was no formal opening. This is postponed until the hospital buildings attached to the school shall be completed. Nevertheless, the gathering which assembled was large and influential, and the attendance of the students satisfactory and encouraging. The visitors were much interested in the arrangements made for the instruction of the students, and the excellently equipped laboratory came in for a large share of well merited praise. It is spacious, comfortable, well lighted, and fitted up with every modern appliance necessary for the work in hand. There can be no doubt that with the special facilities afforded in this laboratory for the practical study of the parasitic agents which play such an extensive rôle in tropical diseases, the future practitioner of the tropics who avails himself of the opportunities here placed before him will occupy a position at the commencement of his career which none of his predecessors have ever enjoyed. With the advantages to be derived from a thorough acquaintance of both the theoretical and scientific

aspects of tropical medicine, let us hope that he will not only make a good practitioner in his tropical home, but also that he will endeavour to advance scientific medicine by seriously attacking some of the many problems that are awaiting solution in those regions, and which it is impossible for others less instructed to attempt.

The lament of most who have hitherto practised in the tropics has been the impossibility of carrying on observations on subjects of the highest importance because of the defects in their early training. There has been no lack of material ready for investigation, but the knowledge of methods has been wanting, and theories have had to take the place of facts. Exceptions only prove the rule. The work of Manson and others has been carried on under peculiar difficulties, and the results form but a microscopic portion of that immense mass of scientific truths which has been and is still waiting to be revealed. The impetus of research, strong in general medicine, has happily been extended to tropical medicine by the efforts of these pioneers. The result is even visible to-day. Theories are beginning no longer to pass counter for facts, and the impulse is to examine even the most cherished doctrines in the light of observation and experiment. This impulse, however, is impotent without proper direction, and is but likely to lead further astray. For illustration we have only to compare the observations and experiments of Ross on the blood and its relations to malaria before he was trained in modern methods, with those of a later date. In the former case they were misleading, while in the latter they are invaluable and pregnant with the most far-reaching results. No better example could be given of the necessity and of the advantage of a practical training in the subjects which will be dealt with in the Tropical School. In the tropics, as elsewhere, the most approved and best methods have to be employed if the best results are to be obtained. These are to be learnt at the school. The syllabus extends over a wide range of subjects, affording the students who work systematically at the school a thorough insight into the diseases which they are likely to be called upon to treat. Opened under

favourable auspices, everything connected with the school bids fair for its future success. That which was but a project a short time ago has become an accomplished fact. All concerned in the movement have good reason to be congratulated. The Seamen's Society is especially to be congratulated on its efforts being crowned with success. A special meed of praise is due to Mr. Michelli, the secretary, whose indefatigable labours have in no small measure contributed to the success of a scheme which was promoted in the Colonial Office and had as its support the powerful influence of Mr. Chamberlain. We trust that in time the School will develop, as it should, into an imperial centre of scientific learning in tropical medicine worthy of the empire.

Article for Discussion.

THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS. ACUTE RHEUMATISM. CHRONIC RHEUMATISM.

THE question started in these columns some four months ago in connection with the comparative frequency of diseases of temperate climates in the tropics, dealt with scarlet fever. Numerous responses have been forwarded in connection with the subject and published in the Journal.

I would now submit the question of rheumatic fever (acute rheumatism).

Rheumatic fever I have never seen in any hospital in China, India, or Egypt, in either natives or Europeans. This may be a faulty observation on my part, but I only state what I observed whilst on a tour in India, during which I visited hospitals in Madura, Madras, Bombay, Quetta, Lahore, Delhi and Calcutta. In Egypt I never met with a case of acute rheumatism, nor in Hong Kong or South China during a nine years' residence. This experience cannot be accidental, nor do I expect it is exceptional. From time to time statements of the absence of rheumatism have appeared; and in the *Indian Medical Gazette*,

December, 1896, Dr. J. Tertius Clarke draws attention to the subject.

Rheumatic pains in fever are of course not referred to, nor are the aches and pains which seem to harass natives, whatever the ailment. The question refers to acute rheumatism and the more chronic forms of rheumatism as understood in England. The sequelæ of rheumatism as witnessed clinically and in the *post-mortem* room, serve to bear testimony in elucidation of this question, but my experience, although clinically sufficient, is too limited as regards the morbid anatomy of the disease to be of value; so far as my experience goes, however, cardiac lesions of the nature of the sequelæ of rheumatism I have never seen *post mortem* in natives of warm climates.

Another phase of the question is:—Are persons who have suffered from an attack of rheumatic fever in Britain more or less liable to a repetition of the attack in warm climates? I can only speak from a very limited experience, and most practitioners in the tropics will have the same difficulty, in obtaining cases to report from, as I had. The cause is this: The majority of persons before proceeding to the tropics are either officially inspected in regard to their health, or voluntarily seek medical advice as to their fitness. Now a person the subject of the pericardial or the endocardial lesions, so apt to follow upon acute rheumatism, are not likely to be "passed," or advised to go to the tropics. The number of old rheumatic fever patients met with in tropical practice is consequently extremely limited. I know, however, of one man who had rheumatic fever in England twice, who dwelt in the moist, hot, relaxing climate of Hong Kong, and never had a recurrence. I know also of a patient who had had rheumatic fever in England and pronounced pericarditis with resulting adhesions, who had no recurrence of rheumatism during a twelve years' residence in North and South China.

These are too few to argue from; but in any case the number is limited, and it is important to accumulate clinical experience in this matter. The question has important practical bearings. Rheumatic fever is so dread a disease in England that were it ascertained that a change to tropical

latitudes was likely to stay its recurrence, an important therapeutic fact would be on the road to be established.

I would urge the readers of this Journal to help these questions forward. It seems a purposeless thing to do to sit down and write that there is "no such disease here." It takes but a moment to do, and it does not require "an article" to be written. This may perhaps be the reason of the hesitation; many a man will take the trouble to write even a lengthy article when a postcard with a single "Yes" or "No" is neglected.

J. C.

British Medical Association.

SECTION OF TROPICAL DISEASE.

HÆMOGLOBINURIC FEVER AND PALUDISM.

By Surgeon W. H. S. STALKARTT, M.D., R.N.

Blackwater Fever Intractable to Treatment.

BLACKWATER fever has been studied much of late, and for some years its importance among the diseases of the tropics has been impressed on the medical profession. The mortality from this malady in certain regions in which its prevalence may be said almost to prohibit European races residing, and its frequent intractability to treatment with the tendency to relapses, even when removed to a healthy climate, render a study of the disease of no little importance. When once the disease has been suffered from, most are agreed that the patient should leave the country where it was contracted at the earliest opportunity, to escape a relapse and its possible fatal effects. However prominent a place this disease may have attained among tropical maladies, its true nature is still a matter of uncertainty.

Association with Malaria.

Blackwater fever is a disease noted only in unhealthy regions, and the term unhealthy one may restrict, according to the late Dr. Parkes's definitions, as meaning malarious. Outside malarial regions it does not occur, as far as I am aware, unless after exposure to possible infection in such places. It appears, however, to be unknown in some malarious regions. This malady has some resemblances to ordinary malarial fever, and there are medical men of large experience who regard it as originating in paludism. Many reasons, however, seem to militate against this view purely and simply, among which is its relative infrequency taking groups of cases of malarial fever together. Very many cases of the latter indeed may be seen before one of hæmoglobinuria is met with. The disease has been considered an aggravated or pernicious type of malarial fever, and among such its percentage would appear very inconstant. Pamponkis gives 156 cases of hæmoglobinuria noted out of 307 pernicious cases at Athens. During the late Benin Expedition, out of some fifty-four cases of malaria, classified as pernicious, four were noted with hæmoglobinuric symptoms. After this expedition, among the many cases of malarial fever treated at the Royal Naval Hospital, Simon's Town, no case of a hæmoglobinuric type was observed, although it is well known how severely the men

of the expeditionary force suffered from climatic effects. I am indebted for the above notes to Surgeons Acheson and Clayton, Royal Navy. One may incidentally note here that in the above instance there had not been any very protracted stay ashore, owing to the excellent manner in which the operations were completed. Again, in this relation may be noted the infrequency of blackwater fever, even the practical immunity from it among those on board ships stationed on the West Coast and East Coast of Africa. During the period I was on the West Coast of Africa Station, and again, during the two years on the Zambesi and Shiré rivers, in neither instance did blackwater fever occur among the crews, although malarial fever was very rife after expeditions up the Vintang Creek, and the Gambia, and the Great Sharcies rivers in one case, and among residents in British Central Africa in the other.

Geographical Distributions.

In many regions has this malady been noted. It is very prevalent in intertropical Africa; and on portions of the West Coast and of British Central Africa it is now almost proverbially severe. In Sicily, Sardinia, East Africa, tropical America and New Guinea, besides other regions, is it found. Also it has been noted in the Indian Terai. In Australia and many districts of India it does not seem to have been observed.

Symptoms.

The symptomatology of this disease has been so fully described that it is needless to dwell on it at any length—the fever, the rigors, the hæmoglobinuria, with the great destruction of red blood-cells, the jaundice, the green or bilious vomiting, together with pain and tenderness over the liver, headache, restlessness, and feeling of weakness. Also the marked prostration after the attack and tendency to syncope. The fever may at first be indistinguishable from ordinary malaria. The temperature may be 105° or 106° ushering in the hæmoglobinuria. In subsequent attacks the fever runs a lower course. It may be pointed out that this is so, too, in recurrent malarial attacks, and in such, with no great elevation of temperature, a grave issue may result where this continues persistent. The rigors in ordinary malaria are initial, but may be masked. In blackwater fever rigors recur even during the fever. Whether this is always distinctly so seems open to question. Hæmoglobinuria is pathognomonic of this disease. There is no hæmaturia. No blood cells are seen in the urine, or only a few by accidental occurrence. Test reaction for blood pigment is extremely marked. Albumen is present in large quantity. Tube casts, granular or pigment-laden, are abundant in the urine. The urine may vary from port-wine-like to porter-like in appearance. In a severe case the jaundice is peculiarly marked and sudden in occurrence. A patient seen overnight may seem unrecognisable in the morning from deep saffron discoloration of the whole body. This arises from the large amount of free blood pigment or hæmoglobin in the circulation. There is no itching of skin present, as in biliary jaundice. Marked jaundice in acute malarial febrile attack has been observed, and is of ill-omen. In recent textbooks on bilious remittent fever, a pronounced icteric or saffron tint of skin is mentioned as a feature arising from modified hæmoglobin free in the blood or deposited in the derma. In milder forms of blackwater fever a much less degree of jaundice may be found. Green or bilious vomiting is often markedly present. That this occurs in severe types of African malarial fever to almost same degrees my experience testifies, or with retching it is a very distressing symptom. There may be much headache and restlessness, with pains in loins, feeling of weakness and yawning. After the attack prostration may be pronounced, and the patient should not be allowed to get up from the recumbent position. The case may progress favourably, or urine contain less albumen or blood pigment. On the other hand, death may ensue from exhaustion, syncope, coma, hyperpyrexia,

uræmia, or suppression of urine. The last is not common. Relapses may occur at longer or shorter intervals, and even after leaving the country, without fresh infection. Ensuing debility, with anæmia, has to be combated.

Etiology.

The following theories are to be considered: (1) That it is due to malarial infection; (2) that it is due to quinine poisoning in malarial cachectics; (3) that it is a distinct disease; (4) that it arises as far as the hæmoglobinuria is concerned from individual idiosyncrasy as to kidneys in a patient suffering from malarial fever; (5) that it is due to food substances ingested. This last does not seem borne out. I have seen cases of blackwater fever in which quinine was seldom, if ever, taken. To me it seems doubtful if true blackwater fever does occur where quinine precaution has been systematically carried out.

The circumstances which bear strongly against the malarial hypothesis to account for hæmoglobinuric fever are the limited distribution of this malady and its non-occurrence in certain highly malarious regions where *a priori* we should expect to find it if so caused. It has, however, been unobserved notwithstanding bad malarial cachexias found in those places. Its occurrence in persons who are not malarial cachectics and soon after coming to particular districts. This would seem to indicate a disease of *locale*. Certain marked symptoms have been regarded as pathognomonic and distinctive from malaria, namely, the green vomit, the pronounced jaundice, the recurrence of rigors, temperature often not much elevated and even running a subnormal course, the hæmoglobinuria with great destruction of red cells and great subsequent prostration. Also its peculiar recurrence in that blackwater fever always begets itself even in mild attacks and with the same train of symptoms varying only in degree. In some cases the vomiting may not be present and the jaundice scarcely apparent; also the rigors not evident. May the subnormal temperature be rather a secondary effect from blood destruction than a primary feature? The hæmoglobinuria is distinctive. In ordinary malaria the parasites convert hæmoglobin into pigment subsequently deposited in the tissues and converted by liver into bile pigment.

A very strong argument against the malarial hypothesis is that if a person suffering from malarial cachexia be sent from Asia to the West Coast of Africa he will very certainly get blackwater fever, but would not do so as long as he remained in Asia. Again, it has been noted that the melanuria ceases independently of treatment and spontaneously; but may not this be said too of some malarial attacks (as of other diseases) where the natural tendency of the system to health has more than counterbalanced the tendency to disease? The non-amenability of this disease to quinine has been pointed out. To what extent this is so has not been settled definitely.

Dr. Sambon's view probably Correct.

The above argument supports the theory that this malady is a distinct disease. This view has been most ably propounded by Dr. Sambon. That small unpigmented parasites and free pigment granules have been found in the blood of blackwater fever patients, as I have also observed, would indicate the incidental occurrence at least of some malarial infection. Further, there have been found *post-mortem* pathological changes characteristic of malaria. These changes do not, however, appear so pronounced as the marked colouration of the urine would lead one to expect. The relatively small quantity of pigment found in the tissues (Dr. Thin) may be explained owing to that the corpuscles are destroyed or their contents liberated, the hæmoglobin passing free in solution into the circulation, and comparatively little is converted into pigment or deposited in tissues. This, too, would account for the free pigment granules, as I have observed in the blood of patients, being seemingly not in excess of an ordinary severe malarial case.

That quinine is the cause of hæmoglobinuria cannot be held. It would appear as an aggravating cause in some cases. There is no evidence as to an individual idiosyncrasy being the cause of hæmoglobinuria, for alterations in kidneys are a secondary condition.

To conclude: As far as our knowledge at present goes, it would seem that:—

(1) Blackwater fever is only found associated with malaria. The circumstances under which it occurs, or which appear to be propagating factors would indicate this.

(2) Blackwater fever is not malaria simply, but a distinct disease, possibly malarial in nature, due probably to an organism having a specific action, or one of peculiar intensity, in giving rise to great blood destruction.

(3) Quinine is not the cause of true blackwater fever, although in certain cases it may aggravate it.

(4) Quinine has been distinctly beneficial in this malady—to what extent is uncertain, but with mixed malarial infection, or in warding off subsequent malaria, it should be given a foremost place. Few, indeed, would care to omit it from their medicinal equipment.

Treatment.

In all cases at all marked the patient should be kept at rest and in a recumbent posture. The usual routine remedies, as a saline to free the primæ viæ, diaphoretics, &c., may be given with advantage; pilocarpin hypodermically, antipyretics, and cool sponging done carefully, as indicated. Quinine, 20 gr. twice at least in the day, should not be omitted at first. Symptomatic treatment as requisite. Draughts of hot water for the vomiting. This if rejected and repeated, relieves stomach of bile and eases retching; also aids diaphoresis. Hydrochloric acid is recommended for vomiting by some, also liq. hydrarg. perchloridi given internally. Morphine hypodermically may be necessary to relieve retching. Quinine hypodermically may be considered. Sulphonal is useful for restlessness ensuing. Bland drinks may be given; milk, and with eggs, if able to be taken. A little stimulant, as the best champagne or brandy, may be necessary. Oxygen inhalation has been suggested as a possible benefit. Weakness or tendency to syncope must be kept in mind during convalescence. Removal to healthy climate should be impressed on patient after blackwater fever, or non-return for at least six months or a year. Pig's bile has been praised as beneficial in the treatment of this disease. Avoidance of chills, sun exposure, wettings, &c., with subsequent prophylactic use of quinine, should be kept in mind.

Discussion.

Dr. SAMBON said that he believed blackwater fever to be a specific disease. Its peculiar geographical distribution and seasonable prevalence, its recent appearance in malarious regions in which it was previously unknown, its characteristic symptoms always constantly the same in the mildest and gravest cases, proved that the specific agent of blackwater fever could not be any one of the several malarial protozoa already differentiated. The closest analogies were not between blackwater fever and tertian fever, or summer-autumn fever, but between blackwater fever and Texas fever or redwater fever which was the hæmoglobinuric fever of cattle. The parasites of tertian fever and those of summer-autumn fever had been found in cases of blackwater fever; but this *per se* was no proof that they were the cause of hæmoglobinuria. That they should be found in a patient suffering from blackwater fever was only what might be expected, because blackwater fever was in many places co-endemic with tertian and with summer-autumn fever. A mixed infection of blackwater fever and summer-autumn fever was therefore just as possible as a mixed infection of tertian and quartan fevers. Several authors had observed a small unpigmented parasite in cases of blackwater fever which, so far as description went, seemed to correspond entirely with the early forms of that found by Smith and Kilborne in Texas fever. The morpho-

logical differences between the parasites of Texas fever and those of the summer-autumn group were so slight, and the concurrence of some form or other of malarial infection with that of blackwater fever was so frequent, that it was not surprising that the specific parasite has not been as yet definitely isolated. No multiple, rosette-like division had been witnessed in Texas fever, as in ordinary malarial fevers, but probably simple binary fission was the mode of multiplication. Possibly in blackwater fever there was the same mode of division. In fact, the association of two parasites within the same corpuscle and their apparent connection had been noticed by Woldert and others. This would be an important diagnostic character if it were confirmed.

Dr. MANSON classified the arguments for and against the hypothesis which would regard blackwater fever as a malarial disease, as follows:—

For,

- (a) It occurred in malarial—very malarial—districts.
- (b) The malarial parasite was often found in the blood.
- (c) It occurred in people who had had many attacks of malaria.
- (d) It occurred only after prolonged residence and consequent malarial saturation.

Against.

- (a) Its geographical range was limited as compared to malaria.
- (b) The malarial parasites were not always found, and when found they were not always of the same kind.
- (c) It was not certain that the fevers preceding attacks of blackwater fever were really all of them malarial in character. It was possible they were, so to speak, incomplete or abortive hæmoglobinuric fever before the symptom from which the disease receives its name was developed—a not unusual occurrence in other specific diseases.
- (d) Cases have occurred within a short period of arrival in the endemic areas of the disease.
- (e) The epidemic seasons of malaria and blackwater fever did not always correspond.
- (f) Blackwater fever was not amenable to quinine.

Professor McLEOD stated that, although acquainted in India with cases resembling blackwater fever, in which the symptoms developed as a sequence to malarial fevers, such cases were not to be confounded with the true blackwater fever met with in Africa.

Major G. M. GILES, I.M.S., desired to emphasise a point which had already been alluded to by Professor Kenneth McLeod. He supposed that everybody had met with stray cases of undoubted malarial fever in which this symptom of hæmoglobinuria appeared. The point was that those cases were so rare in India that no one thought of calling them blackwater fever till so much had been heard of the disease from Africa. Further, cases of malaria complicated in this way met with in India were not even exceptionally fatal, whereas the African disease was an absolute terror. It therefore appeared probable that the apparent resemblance did not extend beyond the striking and alarming symptom common to both conditions, and that the diseases were really quite distinct.

Dr. RHO (Rome) said that Dr. Sambon's theory of the etiology of blackwater fever was certainly the most satisfactory. His theory explained all the symptoms of the disease, and gave a plausible reason for all epidemiological facts; moreover, this theory had in its favour all the arguments from analogy drawn from the hæmoglobinuria of cattle, or Texas fever. If the non-pigmented parasite suggested by Dr. Sambon were to be found in patients suffering from blackwater fever, he would have done a great service to the scientific world. As to the connection of quinine intoxication with blackwater fever, Italian observers had ascertained that in malarial subjects a few centigrams of quinine might give rise to an accession of fever with hæmoglobinuria, very similar to the symptoms produced by pirodin and other blood poisons which destroyed blood corpuscles. These

cases could be explained by Sambon's theory only by supposing that 10 or 12 centigrams of quinine could favour the pathogenic action of his suggested parasite.

Mr. D. C. REES, M.R.C.S., L.R.C.P. (London) said he was disinclined to believe that blackwater fever was malarial. It certainly was not an intense form of malaria, for he had seen cases of blackwater fever of the mildest description, with the passage of cherry-red urine perhaps on two occasions only, and with few other clinical symptoms. It was not chronic malarial poisoning, for he knew of patients who had contracted blackwater fever within two or three months of arrival in Africa. It had been said that there was little evidence of blood destruction, but he believed that there was no disease in which this was so marked. Hæmoglobin could be seen abundantly, both macroscopically and microscopically in the spleen and liver. He could endorse what Dr. Manson had said as to the seasonal difference of blackwater fever and malarial fever. This was most marked at one station on the Niger, where as the malarial curve decreased, the blackwater fever curve gradually increased. All his experience was against the idea that hæmoglobinuric fever was due to quinine poisoning. Quinine was taken largely in Nigeria prophylactically, and those who adhered to this most carefully escaped blackwater fever.

Captain W. T. MOULD, R.A.M.C., mentioned that a patient under his care in hospital for a sprained ankle developed blackwater fever. He had had no quinine for three weeks and no malaria.

Mr. A. P. PARKER, M.B., stated that he had seen cases of blackwater fever in Liverpool from the West Coast of Africa. He would ask whether the fevers met with in Colon, Jamaica, Honduras, and Brazil were not of the same nature. He regarded blackwater fever not as a distinct disease, but merely as the name of a symptom.

The PRESIDENT (Dr. Thin) in summing up the discussion, remarked that in addition to the case which he had recently published in the *British Medical Journal*, 1899, in which a man who died of blackwater fever was found to have the parasite of malaria in the blood vessels of his brain, and pigment in the spleen and liver, he had examined portions of liver and spleen from another fatal case of blackwater fever, and found much pigment deposit in these organs—sufficient to show that at the time of the man's death he had been suffering from an acute malarial attack.

Translations.

BLACKWATER FEVER (HÆMOGLOBINURIA).

By R. KOCH.

Translated by P. FALCKE.

(Continued from page 335, July number, 1899.)

In order to illustrate these conditions, I submit a few histories of the disease:—

No. 1.—Has been in East Africa several years, mostly on the coast. He has had fever a few times, and been treated successfully by quinine; the last attack took place six months previously. Hitherto he has had no attack of blackwater fever. He felt unwell for a few days, had headache, loss of appetite, general weakness; feared an attack of fever, so took fifteen grains of quinine. Three hours after had ague, vomiting, severe hæmoglobinuria, and icterus on the following day. When he came under my care the line of treatment I adopted was to deal with symptoms as they arose. No quinine was given, and the disease ran a mild course, followed

by a speedy convalescence. No relapse occurred during the four subsequent months he was under observation. In this case all the blood examinations were negative.

No. 2.—Has been in tropical Africa for seven years, most of the time in the interior. During this time has only suffered with malaria five times. Six months ago had an attack of blackwater fever. Since then has been healthy. After a march of seven weeks came to the coast in the best of health and looking well. Six days afterwards the patient began to feel ill about midday; feels a dragging and heaviness in his limbs, lost his appetite, and had nausea and a feeling of feverishness. The thermometer registered 40° C. bodily temperature. Soon perspiration set in. In the evening temperature still 39° C. Had a good night, and felt well in the morning. Patient was able to attend to business. At about 11 next day the indisposition recurred, so that the patient was obliged to go home. Here he was seized with violent rigors, lasting nearly an hour, coupled with dry heat. The urine passed soon after was very dark red, and had the appearance of pure blood. He vomited several times. During the same day the icteric colour commenced, and this became more pronounced up to next day. The secretion of bloody urine was considerable, and in consequence of the same there was great anæmia, and a slow convalescence. Treatment symptomatic without quinine. All blood examinations negative. No relapse. Unfortunately, in this case there is no proof as to whether patient took quinine before the attack or not, as at that time the effect of quinine had not been considered. It is, however, quite probable that the patient, according to the custom of Europeans living in East Africa, feeling unwell, had recourse to quinine.

No. 3.—Has been four years in East Africa, and during that time has been under treatment fourteen times for malaria. Two years ago he had an attack of blackwater fever. The patient had his last attack of fever four months ago, from which he completely recovered. One day he felt unwell, and, in order to prevent the attack he considered imminent, he took fifteen grains of quinine. This was followed a few hours after by rigor and subsequent heat. It occurred to the patient that the urine he passed during this attack appeared dark, and he called the attention of the person who was looking after him to the fact. The next morning also the urine had a muddy, brownish appearance. The patient felt weak, but otherwise well. Fearing that the fever would recur, he again took fifteen grains of quinine, and a short time after had a very long and severe rigor. Bad headache and giddiness; frequent vomiting and unconsciousness. The patient could not be admitted to the hospital till evening, when he passed dark reddish-black urine copiously. Icterus also developed next day. The treatment was symptomatic, without quinine. Notwithstanding the considerable loss of blood, the convalescence was undisturbed and fairly quick.

Collective blood examinations negative. No relapse.

No. 4.—Goanese, who nine months previously had come to East Africa *via* Bombay. In India he had suffered much from fever, but had never had blackwater fever. Latterly he had had frequent attacks of fever which were combated with quinine. Feeling

unwell a few days previously, the patient took fifteen grains of quinine. A few hours later rigor, bloody urine, vomiting, and great weakness. He was taken to the hospital in the evening. He had a high temperature, which, however, by next morning had decreased to normal. Icterus could not be confirmed in consequence of the dark skin.

Symptomatic treatment *without* quinine. Favourable course and quick convalescence. Collective blood examinations negative. No relapse.

(To be continued.)

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

TRACHOMA AND RACE.—A very important and interesting communication from Dr. J. Renner, of Sierra Leone, appeared in the *British Medical Journal* of September 16, in response to my appeal for information from the West Coast of Africa on the subject of the incidence of trachoma. Dr. Renner, who has practised for seventeen years in Sierra Leone, fully endorses the truth of what I believe may be considered an axiom, viz., that trachoma is a disease of "race, not place," and is also in entire accord with American ophthalmic surgeons as to the immunity of the negro race—an immunity he believes to be absolute. The facts he adduces in support of his belief are very striking, and form a complete body of evidence relating to all classes of the negro population—indigenous adults, indigenous children, adults and children from the different West Indian Islands residing in the colony.

(1) *Adults*.—I quote the evidence *in extenso*: the names of the different tribes should form a most useful record for future reference.

"As surgeon in charge of the Colonial Gaol at Freetown, which has an average of 230 prisoners, who come from the labouring classes, of nearly all sections of the heterogeneous tribes from the population of this colony and its Protectorate, I can affirm that there is not a single trace of this disease noticeable amongst the inmates of the prison. The nationality of the prisoners may be classified as follows:—

Nationality	No.	Nationality	No.
Konnoh	1	Eboes	5
Aakuhs	18	Kroos	2
Mendis	57	Mokoe or Calabar..	1
Sherbroes	33	Baggas	2
Bassas	2	Vays	2
Jolloffs	5	Serrikulis	2
Timinis	55	Limbahs	4
Mandingoes	6	Congos	1
Susus	8	Yalunkas	1
Lokkohs	4	Gallinas	2
Jamaicans	7	Popos	3

These represent the adult section of the population."

(2) *Children*.—Here again the same remarkable immunity is apparent. In the two large negro schools, attended by 200 boys and 125 girls respectively, not a single case of trachoma has ever been observed—a marked contrast to our own Board Schools.

(3) *West Indian Negroes*.—"Amongst the West Indian negroes from the different West Indian Islands, and their children in this colony, the same immunity is observed."

Dr. Renner concludes his valuable communication by expressing his firm conviction that "the negroes of the West Coast enjoy an absolute immunity from trachoma."

I commend this piece of positive evidence to the consideration of Dr. Van Millingen, in the belief that it will very considerably shake his conviction that the incidence

of trachoma is determined solely by sanitary surroundings and has no connection whatever with race.

Dr. Freeland, of Antigua, whose statement that trachoma is common among West Indian negroes I commented on in the August number of the Journal, writes to inform me that he is unable to say to what race or races the negroes he has seen suffering from trachoma belonged, or whether the patients were pure-blooded negroes or not; he adds that entropion is rare. As even a slight admixture with a receptive race destroys the immunity of the negro (witness the negroes of Egypt), and as cicatricial contraction is a very important element in differentiating trachoma from follicular conjunctivitis, Dr. Freeland's statement can hardly be held to invalidate the precise and detailed evidence adduced by Dr. Renner.

M. T. YARR.

INDIA.

INOCULATION OF AN ENTIRE COMMUNITY WITH HAFKINE'S PLAGUE VACCINE. BY C. H. BENNETT, M.D., Lt.-Col., I.M.S., 26th Regiment Madras Infantry, and W. B. BANNERMAN, M.D., Major, I.M.S., Deputy Sanitary Commissioner, Madras.

Up to the present time no demonstration has been forthcoming of the effect produced on a self-contained community, such as a regiment, by wholesale inoculation with Haffkine's plague prophylactic, so the following description of what happened in one such case may be of interest. The account now about to be given derives additional interest from the fact that the individuals concerned were under close observation during the period referred to, thus rendering the record unusually accurate.

Plague cases began to be reported in Belgaum (a town of 40,700 inhabitants) in October, 1897, five deaths occurring in that month, 111 in November, 156 in December, and 226 in January, after which the epidemic rapidly subsided to 50 in February, and an insignificant number in the following months, till in May it ceased. The 26th Regiment of Madras Infantry were stationed in their lines close to the cantonment and city during this time, and suffered severely. The first reported case among them took place on November 12, when Sepoy 2,224, Govindaswami, was brought to hospital, and died the same day. Next day another sepoy was attacked and died. On the 15th a drummer was attacked, and on the 17th the disease appeared among the followers. By the 21st thirteen attacks had been reported from the lines, four of these being among sepoys. On November 22 B Company went into camp, followed on the 23rd by C and G, and portions of A, D, and F Companies. By the 28th the whole regiment was in camp, and the lines were being disinfected by perchloride of mercury solution, whitewashing, and removal of tiles from the roofs. During this transition period fifteen more attacks took place, six being among sepoys. In the ten days following complete removal to camp, thirteen sepoys and twenty among families and followers were attacked. Then, as was to be expected, the cases gradually became fewer, and ceased by the end of the year. During this time (November 12 to December 31, 1897) the following had occurred:—

Among sepoys	...	34 cases with 22 deaths =	64·7 per cent.
Among women	...	20 " " 10 " =	50·0 "
Among children	...	16 " " 9 " =	56·25 "
Among followers	...	8 " " 8 " =	100·0 "
Total	78	" " 49	62·8

Inoculation was begun in the camps on December 24, 1897. Little difficulty was experienced in persuading the men to consent to be operated on when it was explained to them that they would be free to return to their lines after inoculation. The example set by General Rollond, the officer commanding, and the medical officer, who were operated on in front of the men, no doubt also helped to remove prejudice, and 229 sepoys were inoculated during the

morning. This was practically the whole of the men off duty that day in the Hindalgi camp. Next day the men who had been on duty the previous day were operated on, and all those without wives or families allowed to return to the lines. Those in other camps and the families were speedily inoculated, and were then permitted to return also. The return was complete by December 30, 1897. A few more inoculations continued to be done up to January 6 among followers, children, &c., but by the end of the year they may be said to have been complete. The total operated on was 1,665, out of a population of 1,746 living in the lines at that date. The 81 not operated on were infants, women far advanced in pregnancy, and the sick in hospital chiefly, though one solitary sepoy has, up to the present time, refused to submit to operation. After this date two cases occurred in January; both had been inoculated, and both recovered. No cases were reported for the next six months, though, as we have shown above, the epidemic was at its height in the city and cantonment in January. That this practical immunity of the regiment was not due merely to the disinfection of the lines will, we think, be manifest to anyone studying the occurrences during the second epidemic from July to December, 1898, which will now be described. It should here be stated that the men were, after inoculation, allowed to visit the cantonment and city freely up to July 4, 1898.

The second epidemic in Belgaum began in June, reached its height in October, and thereafter declined till January, when it ceased. The table given below shows the numbers of deaths from plague reported in the city and cantonment, month by month, contrasted with the attacks in the regiment:—

Dates.	Deaths reported from City and Cantonment.	Attacks in the Regiment.
June, 1898...	14	0
July, " ...	215	1
August, " ...	304	2
September, " ...	698	2
October, " ...	999	4
November, " ...	275	2
December, " ...	65	1

From the above it is manifest that the numbers attacked in the regiment kept pace exactly with the severity of the epidemic in the neighbouring town, rising and declining with it. We have seen how heavily the regiment suffered during the first epidemic, so their escape at this time requires explanation. The only measures taken by the authorities were placing the cantonment and city "out of bounds" for the troops after July 4, and the disinfection of the few huts that became infected. Both these measures had been taken in the first outbreak, and had proved totally inadequate. How then did the regiment escape during the second outbreak? The men of the regiment were so satisfied with the effect produced by the first inoculation that they made no objection to being reinoculated in August, and this operation was so thoroughly performed that practically no one in the lines was left unprotected. This latter circumstance deprives us of the complete demonstration afforded by the presence of an unprotected population living amongst those protected, but those living on the spot, at least, had no doubt why they escaped. Of the twelve persons attacked during this period three were not inoculated, and they all died; nine had been operated on, and of these three died and six recovered. The history of the three uninoculated persons is interesting. The first case was a sepoy who was not inoculated, because it was supposed he had been attacked by plague during the first epidemic, though from the history this is more than doubtful. The second was the wife of a sepoy who had just joined her husband a few days previous to the development of the disease, and before inoculation could be performed. The third was a European officer, who probably trusted to the

comparative immunity enjoyed by his race during the present outbreak of plague in India.

The above results seem to point to the truth of Haffkine's expressed opinion that in his prophylactic we have a means of controlling an epidemic of plague, and converting it into a manifestation of sporadic cases only.—*Indian Medical Gazette*, June, 1899.

AFRICA.

AN ANALYSIS OF 449 CASES OF MALARIAL FEVER ADMITTED TO THE BARBERTON HOSPITAL DURING A PERIOD OF FIVE YEARS (1893-97). BY WALTER H. HAW, M.R.C.S., BARBERTON.

South African Medical Congress.

Having, by the courtesy of Dr. Powrie, Hospital Surgeon, obtained the temperature charts of the Barberton Hospital from January, 1893 to December, 1897, I have thought that an analysis of the malarial cases admitted during this period may prove of interest, although it is far from complete, on account of lack of notes respecting the individual cases. The temperature charts, with, in some few instances, short notes appended, formed my only guide; but, in spite of these disadvantages, much may be learnt concerning the incidence of malaria in the Barberton District from a study of them. The usual idea of Barberton is that the malaria is deadly there. This may have been the case in earlier years, when the pioneers first made their appearance, and lived their lives of recklessness and dissipation, lives which in too many cases were sacrificed to the malaria fiend, for malaria, alcoholism, and exposure are the allies which wrought such havoc. It is consoling to turn from the recollections of those days to the sober records, however incomplete, of a hospital in the very centre of the district, and the records of which form a focus from which a clear image of the history of malaria may be obtained. It is consoling, I say, to find that out of 449 cases of malaria treated during five years, only 3.14 per cent. succumbed, that is 14 cases, in only five of which no complication is recorded. A record such as this should, in my opinion, dispose for ever of the bad name which Barberton has unhappily acquired through the history of the earlier settlers and prospectors. It is not my intention to describe in any very great detail the results of the analysis; I will merely mention the salient points, and invite particular attention to the tables and charts, which I have prepared. The temperature charts were examined with a view to the elucidation of the following subjects:

- (1) The types of the fevers, and their occurrence.
- (2) The virulence of the poison at different seasons, as shown by the highest average temperature, calculated for five years.
- (3) The death-rate.
- (4) The complications observed.

In all, 449 charts were examined, viz., 147 in 1893, 45 in 1894, 78 in 1895, 75 in 1896, and 104 in 1897; giving a yearly average of 89.8 cases for the whole period. It will be noticed that in 1893 the greatest number of cases were admitted. It was in this year that malaria was so prevalent, not only in this district, but over the whole of the Transvaal, the railway workmen especially suffering severely. It is curious that the following year, 1894, should have been marked by so small a number of admissions. It is also remarkable that the admissions during 1895 and 1896 were nearly equal in number.

The accompanying table shows the admissions for each month of the five years. It also presents several other interesting features. By adding together the number of cases admitted in January, 1893, January, 1894, and so on to January, 1897, we arrive at a total of 22 cases admitted during January, and covering a period of five years. No cases were admitted in January, 1893 and 1894, 9 in 1895, 2 in 1896, and 11 in 1897. By comparing the admissions during the several months in this way, we find the admissions as follows:—

January, 1893—1897, 22 cases	
February ..	84 "
March ..	45 "
April ..	65 "
May ..	86 "
June ..	57 "
July ..	38 "
August ..	18 "
September ..	14 "
October ..	19 "
November ..	25 "
December ..	21 "

444 cases

Giving the following percentages, the curve of which will be seen in the chart below. Five cases are unaccounted for here, the month of admission not appearing on the temperature chart.

January ...	4.95	per cent. for 5 years
February ...	7.8	" "
March ...	10.1	" "
April ...	14.6	" "
May ...	19.8	" "
June ...	12.8	" "
July ...	8.5	" "
August ...	4.05	" "
September...	3.1	" "
October ...	4.2	" "
November...	5.6	" "
December ...	4.7	" "

On consulting the chart, it will be seen at a glance that a steady rise in the wave of malarial incidence begins in January, and reaches its highest point in May, from which time there is a fall to September, after which month a slight rise follows. It is interesting to compare the curve of highest temperatures with this percentage curve of malarial incidence. It is seen that, while the percentage curve rises steadily from January to May, there are two interruptions in the "highest temperature" curve, viz., during February and April. After this latter month the "highest temperature" curve follows the percentage curve very closely.

It is interesting, too, to mark that the percentages of December and January are in close approximation.

The percentage charts for the various years are also, I think, worth perusing. A few points may be remarked upon. The 1893 epidemic seems to have set in with remarkable suddenness, only eight cases being admitted between January 1 and March 31. During April the admissions are more than three times those in March. In May the admissions were 44 out of 147, nearly one-third the total number admitted during the year. This fact accounts in large measure for the great preponderance of the five years' percentage for May, in spite of the fact that there were only two admissions in May, 1894. The November rise in 1893 was more marked than in any of the following years. The year 1894 was the mildest of all, the highest number of admissions being recorded in June (14 cases). A reference to the yearly percentage curves will, perhaps, prove of more benefit than a wordy description, so that I will pass on to a consideration of the types of Barberton fevers as shown by the temperature curves.

Before entering upon this I must draw attention to one or two points. In the first place, no blood examinations were made for diagnostic purposes; secondly, the time of quinine administration is not recorded, and except in quite a few cases, no discrimination is made between first attacks, relapses, or recurrences. These omissions do not, I think, materially interfere with the results of an examination of the types of fever observed.

I have placed the types noted in these 449 cases under the following headings:—

- (1) Quotidians showing a marked daily rise of temperature.
- (2) Simple tertians, showing a rise every other day.

(8) Summer tertians, which may be described as quotidians with a prolonged paroxysm, in the course of which may usually be observed a pseudo-crisis and a pre-critical elevation. There is a return of the fever to normal on the third day.

(4) Continued, sub-continued, and remittent fevers.

(5) Cases showing no type.

(6) Cases with no fever.

In the first class were 246 quotidians, in the second 23 simple tertians, in the third 41 summer tertians, in the fourth 42 continued, in the fifth 42 of no type, and in the sixth class 62 with no rise of temperature, making a total of 446 cases in which the type can be made out.

These figures give the following percentages:—

Quotidians	5.8	per cent.
Simple Tertians	5.2	„ nearly.
Summer Tertians	9.2	„ nearly.
Continued	9.4	„
No Type	9.4	„
No Temperature	14	„

These figures, of course, cover the whole period of five years.

The lowest number of quotidians were admitted during the month of December, and the highest during May, this latter fact being chiefly due to the large number admitted in May, 1898.

Twenty-three simple tertians were admitted during the five years. The occurrence of this form is quite irregular. For example, in 1898 the cases occurred between May and September; in 1894 during June and July; in 1895 between May and October; in 1896 between January and April; and of two cases in 1897 one occurred in March and the other in November.

Of the 41 summer tertians admitted, the greater number occurred in March, April, and May.

With regard to the continued and remittent fever, it may be remarked that January is the only month which was entirely free during the whole period. With this exception, the occurrence of these fevers is quite irregular. Of the 42 cases exhibiting no type, eight occur in July. Of the cases with no rise of temperature during their sojourn in hospital (usually old cachectic cases) it may also be said that their occurrence is more or less irregular, but to a certain extent limited to the first seven months of the year. I have further attempted to estimate the virulence of the malarial poison by means of the average highest temperature for each month and covering the five-year period. *Vide* Chart No. 1. We learn from this that the temperature rises highest in March, April, May, June, and July; during the succeeding months there is a fall of 2°, with a subsequent rise in November again.

The number of deaths registered was 14, giving a death-rate of 3.14 per cent. Most of the deaths took place in May (8 out of 14 cases).

Of these 14 deaths, quotidians account for 5, continued for 4, cases of no type for 4, and summer tertians for 1. In the majority of fatal cases one or other complication was the immediate cause of death. The complications noted were:—Coma, pneumonia, exposure 2 cases, diarrhoea 2 cases, dysentery, miscarriage, cardiac disease, and perforation of bowel. In 4 cases no complication is mentioned. The total number of complications noted in the 449 cases was 85, among which dysentery holds the first place, pneumonia and diarrhoea coming next.—*South African Medical Journal*, July.

TOXIN AND ANTITOXIN.

Stephens and Myers (*Journal of Pathology*, vol. v., 1898), in an extended series of studies on the action of cobra-poison on the blood show in its relations to the general question of immunity that Erlich's chemical theories are more in accord with their facts than those of other writers. They show:—

(1) Cobra-blood delays or inhibits clotting of blood in vitro.

(2) This inhibitory action upon clotting of cobra-poison is neutralised by anti-venomous serum in vitro.

(3) This action of the anti-venomous serum in vitro is specific.

(4) The anti-venomous serum itself, when added to blood, delays clotting.

(5) For certain doses ($\frac{1}{100}$ gr.) the measure of the neutralisation in vitro, using clotting as the test-reaction, is also the measure of the neutralisation in corpore for guinea-pigs.

(6) The neutralisation of the toxin by the antitoxin in vitro is certainly not vital or cellular, but must be chemical.—*Merck's Archives*.

FRANCE.

The suggestion made by Dr. Sambon in the columns of this Journal (vol. i., p. 263), that there is a difference of great diagnostic importance between the reproductive processes of the parasites of ordinary malarial fevers and those of hæmoglobinuric fevers has been confirmed, as far as Texas fever is concerned, by Laveran and Nicolle in the August number of the *Comptes rendus hebdomadaires des séances de la Société de Biologie*. These authors have definitely established the fact that *Pyrosoma bigeminum* multiplies by simple binary divisions within the blood of affected oxen.

News and Notes.

THE OPENING OF THE LONDON SCHOOL OF TROPICAL MEDICINE.

No invitations were issued for the opening of the new school. Dr. Manson's address was delivered to a small gathering consisting of the Committee of the Seamen's Hospital, the Staff of the Hospital, and intending students.

The subjects dealt with by Dr. Manson and the importance of their bearing will, however, find an audience all over the world. The separate points touched upon were appropriate to the occasion, and, in point of public interest and public health, none perhaps more so than the practical way in which he handled the spread of plague by rats. Every one, having the least knowledge of the subject, will agree with him as to the imperative necessity of destroying the rats and mice when a city is plague threatened, that is, *before* a possible outbreak.

Percival A. Nairne, Chairman of the Seamen's Hospital Society, occupied the chair.

Sir Frederick Young proposed that a vote of thanks from those assembled be conveyed to the Right Hon. Joseph Chamberlain for the active part he had taken in founding the London School of Tropical Medicine, and for the financial support he had obtained for it.

The motion was seconded by Mr. G. Lidgett and carried unanimously.

Sir Chas. Gage Brown proposed a vote of thanks to the Seamen's Hospital Society, for the public-spirited way they had taken up and carried through this all-important scheme. Surgeon-General Ross, Principal Medical Officer of British Guiana, seconded the resolution, which was carried unanimously.

The Chairman, in thanking the meeting in behalf of the Seamen's Hospital Society, referred to the services of Mr. P. Michelli, the Secretary, in connection with

the foundation of the school. It is well known to those interested in the School how Mr. Michelli has worked so as to ensure its development and prosperity, and in how able a manner he has steered it through many initial difficulties.

YELLOW FEVER IN THE UNITED STATES.—The severity of the epidemic of yellow fever at Key West and New Orleans shows no signs of abating. On October 9 twenty-four fresh cases of the disease occurred at Key West alone. Although the southern ports of the United States are not infrequently visited by yellow fever, there is a danger that the increased intercourse with Cuba consequent upon its annexation is likely to increase the danger. Investigations are being made concerning the possibility of the transmission of yellow fever by the mosquito bite.

"ON the metamorphosis of the young form of *Filaria Bancrofti*, Cobb [*Filaria sanguinis hominis*, Lewis; *Filaria nocturna*, Manson], in the body of *Culex ciliaris*, Linn., 'House Mosquito' of Australia," by Thos. L. Bancroft, M.B. Edin.

In this paper, read before the Royal Society, N. S. Wales, June 7th, 1899, the metamorphosis of *Filaria nocturna* in the body of the mosquito is shown to take from sixteen to twenty days for its completion, instead of seven days as was thought. Previous observers, endeavouring to follow Manson, were unable to keep their mosquitoes alive sufficiently long. The writer discovered a means by which mosquitoes may be kept alive in suitable glass vessels for upwards of two months; he feeds them on ripe banana. He explains how it occurred that Manson saw the final stage of the metamorphosis occasionally in what he thought were seven days old mosquitoes; these particular mosquitoes had imbibed filarinated blood weeks before the time when they were captured and already contained advanced stages of the metamorphosis. Any one may now easily verify Manson's work by merely placing two or three "house mosquitoes" under the mosquito net curtains of the bed in which a filarious person sleeps, preferably late at night, and when the person is asleep; the next morning the mosquitoes, which have sucked blood, are transferred to a large glass vessel and fed on banana; in twenty days every one of them will contain actively moving filariæ.

We hope to publish this important paper in full.

Communications, Letters, &c., have been received from:—

- A.—Dr. D. E. Anderson (Paris).
- B.—Dr. J. Berry (London).
- C.—Mr. P. T. Carpenter (Punta Gorda); Lieut.-Col. O. H. Channer (Ealing); Dr. J. Tertius Clarke (Perak).
- D.—Dr. A. B. Dalgetty (Madabpore).
- F.—Capt. C. Framside, I.M.S. (Rajahmundi).
- M.—Dr. Wm. A. Murray (Dublin).
- P.—Dr. Fred. Pearse (Calcutta); Dr. A. Plehn (Driezmin).
- V.—Mr. E. D. Vanderburgh (Hainan).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.

Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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- 1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.
- 2.—Manuscripts sent in cannot be returned.
- 3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

BLACKWATER FEVER IN THE NIGER COAST PROTECTORATE.

By A. H. HANLEY, F.R.C.S.I.
District Medical Officer.

THE following cases of blackwater fever that I have met whilst practising in the Niger Coast Protectorate may be interesting, and, I trust, will afford some information to those whose lot it is to practice where this scourge exists. The first case I came across was in June, 1887, after I had been in residence on the Coast five months. At this time blackwater fever was seldom seen in the Protectorate, and was quite unknown to some of the older residents.

CASE I. June 15, 1887.—G., clerk, aged 29, had not been looking well for some time. Yesterday, whilst down with fever, got up and tried to do his work. To-day awoke not feeling well, but insisted on trying to work. At 9 a.m. was seized with well marked rigor. When I saw him at 11.30 a.m. he was just beginning to get warm. Noon, temperature 105°, pain in head, vomiting, looks very pale. Gave quinine, grs. 15, antipyrin, grs. 10. 5 p.m. perspiring freely, temperature 102°, gave quinine, grs. 15.

June 16.—Had rather good night. 9.30 a.m., temperature 100.6°, pulse 88. Face looks very wizened, tongue clean. On passing urine noticed it was the colour of blood. 10 a.m., had a severe rigor. 12.30, temperature 105.4°, pulse 120, head throbbing, eyes dull, face bloodless, vomiting; passed urine again, which had following characteristics: colour of stout, on standing deposited a sediment not unlike ground coffee. On heating flakes began to form, some floating and others sinking. Gave antipyrin 4.45 p.m.,

temperature 103.6°, pulse 108. Face has a well-marked yellowish tinge; passed urine of same character as last; vomiting has stopped; able to take soup. Gave quinine grs. 10. 8 p.m., temperature 103.3°, pulse 96, no pain, restless.

June 17.—Had a good night; 7 a.m., temperature 99.8°, pulse 88. Gave quinine gr. 15. At 8 a.m. had a rigor; passed urine almost the colour of blood. 10 a.m., temperature 104°, pulse 108; has a distinctly jaundiced appearance all over his body. Gave antipyrin grs. 15. Noon, feels easier, temperature 101.2°, very weak, gave nutrient enema. 5 p.m., temperature 104°. 8 p.m., 102°, urine still of a very dark colour; gave nutrient enema.

June 18.—Had bad night, vomiting continuously, great thirst, urine clearing. 7 a.m., temperature normal; called Dr. Parker in consultation. Gave at his suggestion an emetic followed by morphia gr. ½. Two nutrient enemata.

June 19.—Had a restless night; water clearer and he feels better, still unable to retain anything on his stomach; tongue of a yellowish colour.

June 20.—Water passed at 7 a.m. almost clear, a short time afterwards he passed 7 ounces of a blackish green colour; in the evening it was clear again. He continued to improve and was invalided.

CASE II.—C. has been five years on the Coast. Had several attacks of blackwater fever, sent for me, when I found that he had just passed a large quantity of the characteristic blackwater urine. Temperature and pulse normal, no pain. Next day urine clear and remained so.

CASE III. Benin, 1891.—L., aged 28, agent, first voyage to Coast. Has been on Coast a few months, during which time had slight attacks of fever. After exercise on the trapeze was seized with what he called fever and ague. When I saw him at nine p.m. he

was very restless, temperature 103°, vomiting, and had just passed a quantity of the characteristic black-water urine. Gave antipyrin, grs. 20, followed by quinine, grs. 15. Next day his temperature varied between 101° and 102° and his urine began to clear. On the third day his urine was clear and he made an uninterrupted recovery.

CASE IV. *Degema*, February, 1896.—S., aged 29, clerk. First voyage to Coast. Been out a few months.

Feb. 5.—Complained of not being very well, headache, temperature 103°; ordered him to turn in. Gave calomel, grs. 6, followed by quinine, grs. 15.

Feb. 6.—Had a bad night, vomiting, bile very jaundiced; temperature 101°; passed during night the characteristic blackwater urine, no pain. Put him on liq. ammon. acit. co. and spir. etheris. nit. 6 p.m., slight vomiting during day, able to retain nourishment, temperature 100°.

Feb. 7.—6 a.m., had fairly good night, looks worse than yesterday, temperature 98°, pulse weak, passing little urine. Put him on arsenic, strychnine and brandy. Bowels moved during night, cupped and applied mustard over kidneys. Noon temperature 99°. Gave sp. etheris. nit. potassii. cit. and tinct. scillæ. 8.30 p.m., able to take a little nourishment and looks better, urine still scanty, though improved in colour. Heard he was very restless during night and insisted on sitting up on the verandah. After he had been there about ten minutes the person he was talking to noticed he did not reply and found he was dead.

CASE V. *Degema*, February, 1896.—T., aged 24, clerk. Strong, healthy looking man. First voyage. Been on Coast about two years, during which time has had several attacks of fever.

Feb. 14.—Fever in afternoon.

Feb. 15.—At work during the day. Went down with fever at 5.30 p.m. 6.30 p.m., temperature 102°, gave phenacetin, grs. 10.

Feb. 16.—6.30 a.m.—Had a bad night, very restless with bilious vomiting, temperature 99.4°, pulse 120. 8 a.m., gave quinine, grs. 12, and champagne, and the same at noon. 4 p.m., got up to go to stool but fainted; his motion passed away whilst in that state; when I saw him immediately afterwards, temperature 101.4°, pulse 130.

Feb. 17.—Had fairly good night, rambling somewhat. 6.30 a.m., temperature 99°, pulse 130; as there was no evidence of his having voided urine since yesterday I passed a catheter and drew off an ounce of the characteristic blackwater urine. Gave spir. etheris. nit., potassii. cit., liq. ammonia acet. and tinct. digitalis, and put him into a hot mustard bath; within a short time of his removal from bath he passed 15 ounces of very dark-coloured urine. Put him on 1 oz. brandy every three hours; noticed when I was putting him into bath that skin had a distinct yellowish tinge. 11 a.m., vomiting, temperature 102°, pulse very weak. Gave nutrient enema containing tinct. digitalis min. 15, quinine sulph., grs. 10. 1 p.m., passed 15 ozs. of urine. 2 p.m., appears better. 3 p.m., he took and returned two eggs, milk and brandy, still partially delirious and heart very weak. Gave strychnine sulph., grs. $\frac{1}{2}$. 6 p.m., temperature 102.8°, pulse 130. Gave nutrient enema containing

quinine, grs. 20. 8.15 p.m., delirious, temperature 101°, pulse 132. Gave strychnine, grs. $\frac{1}{2}$ and hyosc., grs. $\frac{1}{15}$. About 10 p.m. went to sleep. 1 a.m., breathing normal. 1.30 a.m., respiration rapid, moaning, pupils dilated and staring. He died quietly at 2 a.m. on the 18th.

CASE VI.—*Degema*, February, 1896. M., age 26; clerk; first voyage; been out about two-and-a-half years.

Feb. 23.—Had an attack of fever at 2.30 p.m.; 5 p.m., temperature 102°, pulse 104.; perspiring freely. Gave quinine 10 grs. and soup, and one blue pill with colocynth and hyoscyamus.

Feb. 24.—Felt well in the morning; 8 a.m., temperature normal. Gave quinine 10 grs., and ordered him to remain in the house; he, however, got up and went to work. 5 p.m., temperature normal; noticed conjunctivæ appeared rather yellow. At 8 p.m. he had an attack of fever; 9 p.m., temperature 106°. Cold sponged him with brandy, lime-juice and water, and gave spir. etheris. nit., liq. ammon. acet. and phenacetin. 10.15, temperature 105.6°. As skin was not acting, gave hypodermic of pilocarpine gr. $\frac{1}{4}$. He soon began to perspire, when suddenly his skin became dry and hot again. For next hour temperature varied between 104° and 105°, and pulse between 116 and 120. Respiration hurried; bilious vomiting. 1 a.m., passed 18 ozs. of the characteristic black-water urine, neutral, sp. grav. 1018, loaded with albumen; no bile pigment; is very restless. 1.30 a.m., antipyrin grs. 20. 1.45, perspiring freely and feels easy. 2.15 a.m., temperature 103.2°; quinine grs. 9; slept till daylight.

Feb. 25.—6 a.m., temperature 101.2°, pulse 116; is distinctly jaundiced. 7 a.m., passed 17 ozs. of very dark urine; vomiting. 9 a.m., able to retain nourishment; quinine grs. 20; bowels moved. 6.30 p.m., temperature 102°, pulse 120; vomiting. 7.30 p.m., antipyrin grs. 20. 8.30 p.m., temperature 103°; antipyrin grs. 20. 10 p.m., temperature 100.8°, pulse 120.

Feb. 26.—Had a very restless night; temperature varied between 100° and 103°; gave morphia gr. $\frac{1}{2}$ and atropia; this gave temporary relief; breathing hurried and distressed; cold sponged with brandy, lime-juice and water, which he found very refreshing. 6 a.m., temperature 102°, pulse 112; passed urine less dark-coloured than yesterday; vomiting very distressing and of a greenish colour; gave nutrient enema, which was only retained for an hour. 10 a.m., gave nutrient enema. 2 p.m., gave nutrient enema containing tinc. digitalis mins. 20, quinine grs. 20, which was retained. 3 p.m., temperature 103.8°, pulse 132; cold-sponged. 6.30, temperature 103°, pulse 140; vomiting; gave nutrient enema containing quinine grs. 12, antipyrin grs. 10, tinc. digitalis mins. 15. 8.30 p.m., temperature 101°, pulse 132. 11.30 p.m., very restless and thirsty; vomiting continuously; passed urine which is clearing rapidly; gave hypodermic of morphia and atropia, when he slept till day-light.

Feb. 27.—6 a.m., temperature 101°, pulse 132; gave nutrient enema containing quinine and digitalis. 4 p.m., much improved; appears satisfied that he will recover; temperature 100°, pulse 114; gave nutrient enema; passing normal quantity urine; acid, trace of

albumen, sp. grav. 1011, brownish colour; no bile pigment; vomiting inclined to stop; able to swallow a little arrowroot; gave during day quinine grs. 25.

Feb. 28.—6 a.m., temperature normal, pulse 100. 6 p.m., pulse 102. 8 p.m., pulse 103; gave during day quinine grs. 20.

Feb. 29.—6 a.m., temperature 100°, pulse 104. 4 p.m., temperature normal; gave during day quinine grs. 15.

March 1.—6 a.m., had a good night; still continues to improve; temperature normal, pulse 108; put him on Easton's syrup and discontinued quinine. 5 p.m., temperature 104°; gave quinine grs. 15. 6 p.m., temperature 105°, pulse 128; antipyrin grs. 20. 8 p.m., temperature 103°; quinine grs. 10; no vomiting and not much thirst.

March 2.—6 a.m., temperature normal, pulse 110. 6 p.m., temperature 100°, pulse 112; taking plenty of nourishment; gave quinine grs. 15 during day.

March 3.—Very weak; put him on board a home-ward steamer, and he soon recovered.

CASE VII.—*Brass*, August, 1893. C., aged 27, agent, eight years on coast.

August 25.—Had fever.

August 26.—Saw him for the first time 8.20 a.m. He said he had fever during previous night, temperature 99°, pulse 80, perspiring freely. He had just passed eight ozs. of the characteristic blackwater urine. 10 a.m., had severe rigor. 11.30 a.m., temperature 104°, pulse 130; antipyrin grs. 20. Noon, temperature 103.2°; 12.30 p.m., antipyrin grs. 20. 1 p.m., temperature 102.2°. Gave quinine grs. 16 and applied mustard over kidneys; body has a yellowish tinge, vomiting.

August 27.—Had a good night and feels well this morning; 8.30 a.m., temperature normal. 11 a.m., had rigor, but not so severe as yesterday. Gave quinine, grs. 20. 5 p.m., temperature 101°; gave quinine grs. 20, and put him on acetate of potash and squills. The urine is of the colour of blood and not up to the normal quantity.

August 28.—Had a good night, temperature normal, and remained so during the day; urine clearing and increased in quantity. He made an uninterrupted recovery.

CASE VIII.—*Brass*, April 8, 1894—Same patient as in previous case.

April 8.—Patient dined out on previous evening and had a late night; noticed that the urine he passed in the morning was very high coloured. 3 p.m., when I saw him for the first time, he passed urine the colour of blood, temperature normal and pulse normal, tongue coated, not very weak, vomiting bilious. Gave pulv. jalapæ co. grs. 40, also tinct. scillæ and acetate of potash, which he was unable to retain. 7 p.m., temperature and pulse normal. Gave purgative enema. 9 p.m., puffy under eyes, pulse and temperature normal, feels better. Gave acetate of potash and tinct. scillæ. The urine he passed at 3 p.m. colour like blood; specific gravity 1039; albuminous, no bile pigment; urea 4 per cent.

April 9.—Had a good night, passed 13 ozs. of urine between 9 p.m. and 6 a.m.; colour much improved. Specific gravity 1025, albuminous and contains 2½ per cent. of urea. 6 a.m., temperature and pulse normal. 4 p.m., has passed normal amount

of smoke-coloured urine; able to take nourishment. Gave during day digitalis, squills and acetate of potash.

April 10.—6 a.m., temperature and pulse normal, passing normal amount of urine, smoky, albuminous, specific gravity 1020, urea 2½ per cent.

April 11.—Convalescent, rather weak. Put him on arsenic, iron and strychnine.

April 12.—Allowed him to get up.

CASE IX.—*Degema*, Dec., 1896. D., aged 28, agent, second trip to Coast, remained out two and three-quarter years his first tour, been out fifteen months this voyage. Did not take much care of himself.

Dec. 19.—Had an attack of fever.

Dec. 20.—10 a.m., sent a bottle containing a sample of the urine he had just passed. It was of a reddish colour with transmitted light, and on moving it you could see a greenish hue at the edges. Gmelin's test showed bile pigment. When I saw him he was dressed and about to start to attend a meeting, complained that nothing would remain on his stomach, conjunctivæ deeply jaundiced. Whilst I was trying to dissuade him from leaving the house he had a strong rigor. Temperature 102°. Gave antipyrin, grs. 20. 11 p.m., very restless and vomiting.

Dec. 21, 7 a.m.—Temperature 101°, pulse good, says feels pretty well. Great thirst, which nothing appears to relieve, vomiting bile almost continuously, a ¼ gr. of morphia gave temporary relief, passed urine the same as yesterday. Skin of a yellowish tinge. 7.30 p.m., appears in good spirits. Temperature normal. Vomiting stopped. Gave nutrient enema containing digitalis. 8 p.m., pulse very weak, cardiac sounds barely audible. Gave hypodermic of strychnine and brandy. 10 p.m., complained of the cold, evidently a rigor, he soon became warm and his temperature rose to 106.8°, stripped and sponged with brandy, lime-juice and water; this soon reduced temperature to 102°. Pulse 140, very weak; he became unconscious and died quietly at 1 a.m.—Just before death the temperature rose again.

CASE X.—*Degema*, Nov., 1897. H., engineer, aged 24, arrived on Coast May 11, 1896. Previous health, one attack of fever fourteen days after arrival.

Nov. 24, 8.45 a.m.—Said he had a shivering attack about 3 a.m., felt all right at 6 a.m. and turned to work, but soon had to take to his bed. Temperature 103°; pulse 100. Conjunctivæ and skin of a yellowish colour; passed shortly before I saw him urine colour of blood, acid, very albuminous; specific gravity, 1018. Bowels open. Gave quinine grs. 5, antipyrin grs. 10, and a hot lime drink, perspired freely in half an-hour. 9.45 a.m., temperature 102.6°. Gave quinine, grs. 15. 11 a.m., temperature 101.4°. 1 p.m., quinine, grs. 10; this was not retained. 4 p.m., temperature 103.2°, urine clearer and normal in quantity. Gave quinine grs. 10, phenacetin grs. 5. 5 p.m., quinine, grs. 10, bowels moved during day. 8.30 p.m., feels better, temperature 100°; pulse 90, able to take nourishment.

Nov. 25.—Had fever during night with bilious vomiting. 7 a.m., temperature 100°; pulse 96; water clearer. Gave quinine, grs. 15. 8 a.m., temperature, 101°. Gave phenacetin, grs. 5, and hot

lime drink. 10 a.m., quinine, grs. 10. 1 p.m., temperature 100°. Quinine, grs. 10; able to take nourishment. 9 p.m., temperature 100°; pulse 108; urine almost normal in colour, specific gravity 1024, acid and albuminous; inclined to sleep. Gave quinine, grs. 10.

Nov. 26.—Feels much better. Temperature 100°; pulse 96; urine slightly reddish in colour, acid, albuminous, specific gravity 1024.

Nov. 27.—Convalescent.

CASE XI.—*Degema*, Nov., 1898.—B., aged 28. Been on Coast about five years. Third voyage.

Nov. 19.—I wrote him that, as he was having attacks of fever almost daily, it would be as well if he went for a trip.

Nov. 20.—Received a message that he was very ill. Saw him for the first time at 3.35 p.m. He told me that he had a bad night with fever and vomiting, and that early this morning had a strong rigor. His temperature when I saw him was 104.2°; pulse 110; respirations 32. He passed early in the morning, and again a short time before I saw him, a large quantity of the characteristic black-water urine; has a deeply jaundiced appearance; very weak and restless. Gave at once nutrient enema containing quinine, grs. 20, and brandy, and sponged him down with hot water, lime-juice and brandy: the latter he found very soothing. 6 p.m., temperature 100°; pulse 110; respirations 32. Passed quantity of dark-coloured urine; able to retain a little arrowroot; very restless. Gave hypodermic of morphia and atropia.

Nov. 21.—7 a.m., had a fairly good night. Temperature 101°; pulse 104; respirations 32. Skin appears more deeply jaundiced. Gave a hot sponge down and nutrient enema. Urine still very high-coloured, specific gravity 1026, and deposits a copious sediment. Gave during day, sodii bicarb., sodii salicylas, tinc. digitalis, morphia and hyoscine. Still weak and restless, unable to retain nourishment by mouth. Gave nutrient enemata.

Nov. 22.—Slept till 1.30 a.m. Temperature 100°; pulse 122; respirations 32, sighing. Gave nutrient enema. Urine still very dark. 7 a.m., colour of skin clearing. Specific gravity of urine, 1015. Gave nutrient enema. 8.30 a.m., bowels moved. Hot sponged him twice during day, and applied mustard to epigastrium to relieve vomiting. Able to take spoonfuls of brandy-and-water; also retains a little arrowroot. Gave during day by enema and hypodermically quinine, bi-hydrochlor. grs. 15, tinc. digitalis mins. 20, strychnine gr. $\frac{1}{8}$, and at night morphia gr. $\frac{1}{4}$. 6 p.m., temperature 100°, pulse 120; respirations 28. Nourishment in form of enemata.

Nov. 23.—Had a good night. Urine almost normal. Able to take nourishment by mouth. Pulse improved; respirations 24. Gave sulphonal grs. 20.

Nov. 24.—Is now convalescent though very weak. Urine normal. Sent him for a trip.

CASE XII.—*Degema*, January 1899. S., aged 25. Second trip to Coast. Been out this trip three months.

January 7.—Was in a boat from 9 p.m. to midnight.

January 8.—Saw him this afternoon, said was not very well, and thought must have got a chill on previous evening.

January 11.—Heard he was seriously ill. 7.30 a.m., saw him for the first time. He told me that he had fever on the 9th, two previous days, and was very bad last night. Passed during the night and early this morning a quantity of the characteristic black-water urine; colour of red wine with transmitted light; acid. Specific gravity 1037. Much albumen and heavy precipitate. No bile pigment. Temperature 100.8°, pulse 80; skin has a yellowish tinge. 8 a.m., calomel grs. 3. 9 a.m., had a strong rigor, which lasted half-an-hour. 10 a.m., bowels moved. 1 p.m., temperature 101.8°, pulse 115; feels better, able to take a little nourishment. 4 p.m., passed a few drops of very highly coloured urine; feels well, perspiring freely. Gave liq. ammon. acet. fort. and liq. atropia. No desire to vomit. 6.30 p.m., temperature 100.6°, pulse 100. 8.50 p.m., pulse 112, temperature 101.4°, able to retain little nourishment.

January 12.—1 a.m., slept but little; vomiting; passed 1½ oz. urine the colour of blood; temperature 100.4°. Gave Digitalis, liq. ammon. acet. fort. 2.45 a.m., applied mustard over kidneys. 3.45 a.m., bowels moved. 6.15 a.m., temperature 100°, pulse 99.8, vomiting, the vomit of a yellowish colour. 8 a.m., pulse 100; bowels moved, colour a dark yellow. Continued the digitalis and liq. ammon. acet. 8.40, applied mustard over kidneys. 10 a.m., bowels moved, distinctly bilious in appearance. Surface of body is of a bright yellow; restless; sponged with hot water, brandy and lime juice; took little arrowroot. 12.15 p.m., had a motion, and passed few drops urine. 1.30 p.m., pulse 96, temperature 100°. 3 p.m., passed few drops urine. 4.30 p.m., pulse 92 to 98, somewhat irregular. 8.30, passed few drops urine. 9.10, cupped over kidneys; temperature 100.6°.

January 13.—2.15 a.m., gave hyoscine gr. $\frac{1}{8}$. 6 a.m., passed a few drops urine. Gave gin and soda. 7 a.m., pulse 92, temperature 100.8°, put him in a hot mustard bath. 9 a.m., passed 3 drs. of urine of a brownish colour; this was twice as much as he had passed during previous twelve hours; vomiting. Noon, temperature 100.2°, pulse 90. 4.5 p.m., gave hot mustard bath. 5.30 p.m., as no urine had been passed since morning I passed catheter and drew off one teaspoonful. 7 p.m., respiration 20, pulse 80, skin moist. 9 p.m., temperature 100.2°, pulse 88, respiration 24, gave hot mustard bath, also 3 nutrient enemata during day.

January 14.—2 a.m., pulse 75, temperature 99.6°. Gave hyoscine gr. $\frac{1}{8}$; did not appear to have much effect. 6 a.m., temperature 100.2°, pulse 75. No pain anywhere. Gave spir. etheris nit. and liq. ammon. acet. fort. in hot water. 12.30 p.m., temperature 100.2°, pulse 80. 6.20, temperature 100.4°, pulse 75; gave jaborandi and liq. ammon. acet. fort. 9.55 p.m., feels sleepy; passed about a drachm since morning. Nourishment given by enema.

January 15.—Had a good night; temperature during day 100° to 101.4°, pulse 76 to 92; passed 3 drs. urine, which boiled solid.

Jan. 16.—Had a good night; feels better. Temperature during day, 98.4° to 99.2°; pulse 70 to 72. Passed one ounce of urine; albumen $\frac{1}{4}$.

Jan. 17.—Had a good night. 6 a.m., temperature 98.2°; pulse 70; respirations 17. Passed ten

drachms urine; albumen $\frac{3}{4}$, blood and epithelial casts.

Jan. 18.—Acute attack of diarrhoea. Temperature 98.6° to 99.8°; pulse 72 to 80; respirations 20. Passed two ounces of urine; albumen $\frac{3}{4}$.

Jan. 19.—Had a good night. Temperature 99.6° to 100.6°; pulse 72 to 84. Passed four drachms of urine; albumen $\frac{3}{4}$.

Jan. 20.—Diarrhoea. Weaker. Temperature 100.6° to 101.2°; respirations 24. Urine two ounces, not so smoky in appearance. 11.30 p.m., diarrhoea stopped.

Jan. 21.—Had a bad night, diarrhoea having come on again. Temperature 101° to 101.3°; pulse 81 to 91. Getting weaker. Urine passed, three ounces. 11 p.m., very restless and excited, and thought he was dying, with which opinion I was forced to agree. Hyoscin gr. $\frac{1}{16}$.

Jan. 22.—Quite worn out. 6 a.m., semi-stupor. Pulse 106; respirations sighing. Drew off five and a-half ounces of urine, colour like *café au lait*, acid; specific gravity 1018; albumen almost solid on boiling; blood casts and large number of uric crystals and mucous corpuscles. Hiccough; constant vomiting of a dark greenish substance; tongue getting dry and black; can swallow with difficulty. 5.30, drew off three and a quarter ounces of urine.

Jan. 23.—6 a.m., fairly good night. Lies on his back and takes but little interest in what is going on round him. Pulse 100; temperature 99.4°; respiration sighing. Drew off five ounces same as yesterday. 5 p.m., drew off three ounces. He is now in a stupor. Respiration rapid, finishing up with a sigh every five or six respirations. He continued to sink, and died shortly after midnight.

CASE XIII.—Jan. 5, 1899, 6 a.m., a prisoner, native of Degema, came to dispensary and complained of being cold. Temperature 102°; pulse irregular and difficult to count. Within a short time passed urine the colour of blood; acid; large quantity of albumen; specific gravity 1020. Gave two Livingstone rousers and later quinine, grs. 10. 5 p.m., temperature 104.8°; pulse full and bounding. Quinine, grs. 10, phenacetin, grs. 10.

Jan. 6.—6 a.m., had good night. Temperature 100.4°; pulse weak, irregular and intermittent. Urine normal in colour; no albumen; acid; specific gravity 1016.

Jan. 7.—No fever. Both sounds of heart appear to be reduplicated, the first sounding like "sub'du," very short and soft, the second like "dub-dub," being loud and distinct. Pulse 48.

Jan. 9.—Pulse 44, with same characters as on the 7th. The patient says never had a similar attack.

Remarks.—The above cases are the usual types of blackwater fever one meets with in the Protectorate. Cases II. and VIII. I believe, are forms of paroxysmal hæmoglobinuria, and they would be liable to recur without any further malarial infection. A person who has once had blackwater fever is very liable to an attack of paroxysmal hæmoglobinuria, the attack as often as not being brought on by exposure or a late night. It is somewhat difficult to make a distinction between severe and mild forms of blackwater fever. I have seen cases in which the symptoms of the patient attacked for the first time were so mild as

compared to the severer type, that I have regarded it as a case of simple paroxysmal hæmoglobinuria. Almost all the cases of blackwater fever I have seen have been preceded by an attack of fever, and I believe that it is one of the results of malarial infection. I have found quinine of great service in many cases of blackwater fever and I have never known a case of ordinary malarial fever develop into blackwater, in which the patient was confined to his bed and given 20 to 30 grs. of quinine daily, until he was free from fever for twenty-four hours. Case VI., which I treated with quinine, shows a well marked exacerbation that took place on the day I substituted Easton's syrup for quinine. Cases IV., V., VI. occurred at Degema in the same month out of a population of eleven. They were the only cases of fever that I attended in Degema that month; I am unable to explain their occurrence. Case IX. also occurred at Degema in December of the same year. In this case the patient, who lived freely, had persistent vomiting for some time, which stopped on the evening of the night he died. Within a few hours of his death he expressed himself as feeling better, and if it had not been for the state of his heart I might have agreed with him; he himself at this time suffered no inconvenience beyond a difficulty in breathing. Cases of blackwater fever, but more especially the ordinary malarial fever in which there is well marked cardiac weakness, are not infrequently attended by a sudden rise of temperature. Case XIII. is the first case of the kind that I have seen in a native; the microscopic examination of his blood revealed nothing. I have invariably failed to find the malarial parasite in cases of blackwater fever. In Case XII. I took a preparation of his blood a short time before the second paroxysm and detected the pigmented spherical parasite. In all cases of blackwater fever I find it necessary to constantly note the condition of the patient, especially the condition of his heart and kidneys. I usually commence treatment with a dose of calomel if patient has not already had a purgative. If he is unable to retain anything I soon begin to give nutrient enemata, and at the same time give the drugs I think most suitable. I find the hot sponging relieves the restlessness very much and not infrequently induces sleep. morphia or hyosine will generally give the patient a good night. A diuretic including tinc. digitalis or a hot mustard bath will generally promote the action of the kidneys.

CALABAR SWELLINGS.

By S. W. THOMPSTONE, F.R.C.S.E.

District Medical Officer, Old Calabar.

In the course of six years medical practice in Lower Nigeria, including Old Calabar, I have encountered in three black and in four white men peculiar fugitive swellings about the size of half a goose egg, painless, though somewhat hot, both objectively and subjectively, not pitting on pressure, and usually disappearing in three days. They come suddenly and disappear gradually, and occur in any part of the body. I have never seen more than one at a time, but they may recur every month or two, although at no stated intervals. In one instance, at least, the swellings con-

tinued to recur while the patient was on furlough in England. I have examined the blood in several cases, but without positive result as regards filariæ or other parasites.

I would be much obliged if any of your readers would throw light on the cause and nature of these swellings. I may mention that they are well known to Europeans on the West Coast, and are called "Calabar swellings."

We hope that Dr. Thompson's letter will elicit some information on these curious Calabar swellings, and, meanwhile, we draw attention to some remarks on the same subject made by Dr. Argyll Robertson, in his "Further Note of Case of Filaria Loa," read at the meeting of the Ophthalmological Society, on March 14, 1895. He says:—

"My patient has several times directed my attention to ill-defined swellings under the skin of the forearms a little above the wrists, over the dorsal surface of the radius, more marked generally in the right arm. The surface of the swellings was not quite uniform, but did not give one the idea of being produced by a coiled-up worm. The swellings measured about $\frac{1}{4}$ in. in diameter. They were not painful, but occasioned a feeling of stiffness when the arms were used. The swellings occurred at irregular intervals, and were generally most marked in the mornings. Cold had no influence in dispelling them; on the contrary, the application of cold water on one or two occasions seemed to bring the swelling forward.

"My patient informs me that natives of Calabar, and others resident for a time there, are subject to such swellings in the forearms and wrists, to which the natives apply the term '*ndi-tôt*,' or swelling. These swellings she has only suffered from since her return home."

PHAGOCYTOSIS IN MALARIAL FEVER (QUARTAN).

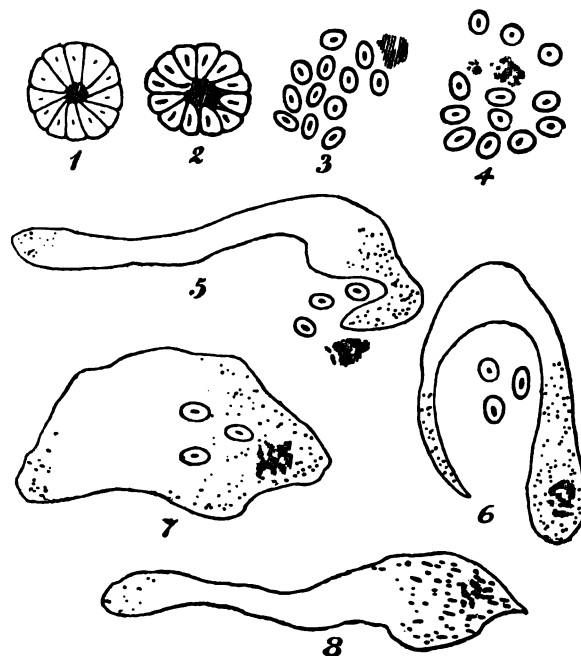
By J. PRESTON MAXWELL, M.B., F.R.C.S.

THE exact rôle that phagocytes play in the destruction of the malarial protozoon has been often discussed, and is mentioned in nearly every work on the subject. At the same time, the treatment of the segmenting forms has not been, so far as I can find, well described anywhere. And this must be my excuse for the description of a process, fascinating in the extreme to behold, and which helps to explain how a quartan fever may be cured by the unaided action of its own active leucocytes.

The subject from whom the blood in question was taken, was a Chinese schoolboy, aged 12. He had had previous attacks of the same fever. On the day in question the cold stage commenced at 7.30 a.m., and he was just passing out of the hot stage when I took the blood at 12 noon.

At 12.30 p.m. I examined the slide and quickly found two rosettes, one being nearly fully formed, the other just beginning to form. Taking the first, at 12.45 p.m., it appeared to be fully formed, and was a most striking object, a faint rim of corpuscles could still be made out around the rosette. At 1 p.m.

rupture took place in one side, and the contents flowed out as if under pressure, shooting out and at once breaking up into separate segmenting forms and a single mass of dark brown pigment. In five minutes' time this single mass of pigment broke up into a larger and smaller piece, and the segmenting forms were beginning to spread.



(1) Rosette at 12.30; (2) at 12.45; (3) at 1, ruptured; (4) two groups (three and nine) of corpuscles; (5) white corpuscles approaching segments; (6) embracing and (7) enclosing them; (8) pigmentary remains of segments.

At this stage several white corpuscles with streaming granules rapidly descended on the segmenting forms. One of the number had previously made an attack on the rosette *in situ*, but had drawn off evidently repelled. It now returned to the attack and was joined by a second, the others, three in number, retiring from the contest. The one (fig. 5) immediately made an attack on and absorbed into its substance the larger pigment mass, and then bending back, as shown in the diagram, wound round the smaller mass of three segments and transferred them into its substance. By 2.10 p.m., that is thirty-five minutes after, all trace of the segmenting forms had disappeared, but the leucocyte was decidedly sluggish, and it was not till twenty-five minutes after that it walked off in search of new pasture. By this time the pigment was beginning to get less in its substance but was still a striking object.

Another leucocyte acted in much the same way and took up into its substance the remaining nine spores. After forty minutes these were still traceable, but in another forty minutes all trace of them had disappeared. The leucocyte at this time showed no inclination for movement. As to the other rosette, it did not mature and break till nearly 4 p.m., and then the same process was gone through again, the segmenting forms being disposed of in the manner before stated.

As to the remains of the blood corpuscle from which the rosette came, its outline was faintly visible for a few minutes and then entirely disappeared.

[The drawings were made from fresh blood, a Zeiss $\frac{1}{2}$ oil immersion being used with eyepiece D.]

Changpoo, 1899.

ON THE METAMORPHOSIS OF THE YOUNG FORM OF *FILARIA BANCROFTI*, COBB [*Filaria sanguinis hominis*, Lewis; *Filaria nocturna*, Manson], IN THE BODY OF *CULEX CILIARIS*, LINN., THE "HOUSE MOSQUITO" OF AUSTRALIA.¹

By THOS. L. BANCROFT, M.B. Edin.

DR. PATRICK MANSON, in a paper read before the Linnean Society of London, March 6, 1884,² remarked:—"Six years ago I described the metamorphosis undergone by the embryo *Filaria sanguinis hominis*, in the body of the mosquito.³ I hoped that (considering the practical importance of a correct knowledge of the life-history of this parasite) the statements I then made would, long ere this time, have been thoroughly confuted or confirmed. . . . With the exception of Lewis in India, Myers in Formosa, and Sonsino in Egypt, I do not know that anyone has worked seriously at the subject. And although both Lewis and Sonsino have confirmed my statements as to the entrance of the *Filaria* into the mosquito, and followed up part of the metamorphosis, neither of them has advanced his observations so far as to be able to confirm my statements as to the later stages of this, or positively to prove that the mosquito is or is not, the intermediary host. Some eminent helminthologists in England accept my statements and endorse the inferences I have drawn—Cobbold, for example. But in other quarters, so far from securing acceptance of my theory, the work of Lewis, on account of the hesitation and scientific caution with which he expresses himself, has had the effect of inducing a certain amount of scepticism. Leuckart is sceptical; and of course the scepticism of so eminent an authority is of great weight in influencing opinion, especially in Germany."

In answer to an inquiry from me as to whether there was any recent work on the subject of "filarial metamorphosis," Dr. Manson wrote, November 15, 1898:—"So far as I know, nothing has been done in 'filarial metamorphosis' since my Linnean Society's paper. Lewis did not go very far with the work. There is an excellent opportunity for work on this subject, and were I in your place, I should certainly go on with it."

In writing to Dr. Manson, I had mentioned the circumstance of my being able to verify his "filarial metamorphosis," but that I had never seen the "actively moving filaria," which he stated left the mosquito's body and lived a free life in water until transferred to the human host.

To this he replied in these words:—"I have seen the 'actively moving filaria' in the seven days' old mosquito a good many times; I used to be able to pick out the mosquitoes containing it. Their thoraces looked plump and juicy to the eye. Of course, you must have hundreds of mosquitoes from which to select such."

Now, in my former investigation there was no difficulty in finding the early stages of the metamorphosis in every mosquito (*Culex ciliaris*) without exception, which had imbibed filarinated blood; those mosquitoes which lived seven days—and none ever lived longer—never contained any actively moving filariæ.

It were useless to make further search for this "actively moving filaria"; either Manson must be in error, I thought, or the *Culex ciliaris* was not an efficient host.

The recent work in India on the metamorphosis of the malarial parasite induced me to study the habits of these insects in this district. I found that I could keep certain kinds of mosquito, particularly *Culex ciliaris*, and a large black species hitherto undescribed, alive in confinement for about two months; one individual actually lived seventy days. Banana was found to be a good food for them. It was ascertained that unimpregnated mosquitoes lived the longest; those that had been impregnated lived two or three weeks, whilst the males rarely lived a fortnight. In my former investigation into filarial metamorphosis, it never occurred to me to feed my filarinated mosquitoes whilst in confinement, accepting the common belief of their only feeding once and dying within a week. Manson, Lewis and Myers, who had worked at the subject, never fed the mosquitoes, and it never dawned upon me that my mosquitoes were dying from starvation.

Having discovered that mosquitoes could be kept alive for long periods in confinement if fed on banana, I was anxious to ascertain what would become of the filariæ which were to be seen in mosquitoes that lived seven days; would they go on developing if the mosquitoes lived longer?

Unfortunately E. S., the filarinated subject, a girl of sixteen, from whom I had obtained filariæ, had left the district, having secured a situation as domestic servant; she was the only person affected with filariasis I knew of.

Dr. Manson's encouragement and a grant of £7 from the Queensland Branch of the British Medical Association to defray the cost of E. S. returning and living with her parents for three months and submitting to be bitten by mosquitoes, induced me to enter upon a fresh investigation on February 1, 1899. It was found that the actively moving filariæ were to be seen, but not before the sixteenth or seventeenth day, sometimes in cold weather not until twenty days, and that no further development occurred in them even after a sojourn of sixty days in the mosquito's thorax.

The final stage of the metamorphosis, i.e., the preliminary alternation of generations, is attained in sixteen or seventeen days; the young filariæ are then $\frac{1}{15}$ " in length by $\frac{1}{150}$ " in breadth, some only $\frac{1}{15}$ " \times $\frac{1}{150}$ "; there is no apparent difference, except in size; there is a well marked intestine with cesophageal bulb, also some differentiation of the body protoplasm into reproductive organs (ovary and testicle), but I have not been able to make out any sexual difference.

The young filariæ are generally only to be found in the thorax, yet a few occur in some instances in the abdominal cavity. There are usually three or four filariæ present, sometimes as many as twenty-five. In twenty filarinated mosquitoes that were killed and examined between sixteen and sixty days, every one of them contained actively moving filariæ.

Mosquitoes bearing filariæ do not appear to be injured seriously; one that was killed fifty days after its meal of blood contained eleven filariæ in the thorax and two in the abdominal cavity.

In mosquitoes fed on non-filarinated blood no filariæ could be detected.

When the mosquito's thorax is torn across several times with dissecting needles in a watch-glass containing water on the stage of a dissecting microscope, the filariæ are liberated and sink to the bottom; they can be seen fairly easily with the naked eye and by aid of a needle picked out; they cannot swim nor move away from the spot where they happen to sink, yet they twist and wriggle about in a violent manner; by means of what appear to be caudal suckers some of them stick to the glass, also to the dissecting needle when touched by the same.

Water is injurious to them, for after three or four hours therein they die. Water, therefore, cannot be the medium,

¹ Read before the Royal Society of N. S. Wales, June 7, 1899.

² *Trans. Linn. Soc. Lond.*, vol. ii., part x., Zoology, p. 367.

³ *Proc. Linn. Soc.*, March 7, 1878. China Customs Medical Reports, Sept., 1877.

as was generally supposed, by which they ultimately reach the human subject.

Directly after having seen the first "actively moving filariæ" wriggling about in water for a couple of hours, I concluded that water was the medium, and wrote a letter to the Editor of the *Australasian Medical Gazette*¹ to that effect, being anxious to correct a former statement² of mine to the effect that the young filariæ died in water [as subsequent observation has shown, that statement did not require correction]. Shortly after having written the letter I found the young filariæ were dead, but concluded that they must have been injured by the cyanide of potassium by which the mosquito was killed. Many experiments were afterwards made with filariæ from mosquitoes that had been dissected whilst alive to insure no injury to the filariæ they contained; it made no difference however, for the young filariæ always died after three or four hours' immersion in water. In mosquitoes that had died a natural death, when examined twenty-four hours afterwards, the filariæ were dead; this occurred whether the mosquito died in water or not. The filariæ never escape naturally from the mosquito's body.

In order, therefore, for the young filariæ to reach the human subject, it would appear that the mosquito must be swallowed. It is not uncommon to meet people who have accidentally swallowed one of these insects, and it seems possible enough that such might occur, especially in those who sleep with the mouth open. In the act of killing mosquitoes with the hand their bodies are ruptured and any young filariæ that might be present, would be extruded on to the fingers and afterwards transferred to the mouth. Mosquitoes when aged frequently get bogged in jam and honey, and by such food it is possible, although somewhat improbable, they could gain entrance into the human stomach. To be infected some may imagine that there must be an easier way than by swallowing mosquitoes; they must remember, however, that Nature has not ordained that the life-cycle of entozoal parasites shall be easily attained; obviously for the reason that, were it easily accomplished, gross infection would occur causing the death of the host and with him the parasites.

Leuckart in his work³ makes the following reference to Manson's discovery, p. 64, footnote:—"From the observations of Manson⁴ there can no longer be any doubt that the few embryos which can pass without danger to themselves through the intestine of the mosquito undergo further development in the body-cavity, in consequence of which they now differ in size and in the structure of the mouth parts from the embryo at an earlier stage. Manson is of opinion that embryos, having thus reached a certain stage in the body-cavity, get into water only on the death of the host, and that they are taken into the human body with the water. This statement still requires demonstration, but even were this proof forthcoming there would yet remain a possibility that the embryos evacuated with the urine (which probably no more represent a useless production than the eggs of intestinal worms which pass out with the fæces) may be transported to certain small hosts, and by these means human beings may perhaps be infected more commonly than in the way pointed out by Manson."

From these remarks it would appear that Leuckart imagined that it was a normal occurrence for embryo filariæ to pass out of the body with the urine; such is not so, however, and is by no means common in those affected with filariasis; it occurs in cases only when there is rupture of a lymphatic or blood vessel in the kidney or bladder; the filariæ when mixed with urine are rapidly altered by endosmosis or exosmosis and live but a short time. The same applies to dogs affected by the *Filaria immitis* in which however it is even of rarer occurrence.

How did it come about that Manson saw the final stage of the metamorphosis in mosquitoes seven days old?

This I believe to be the explanation:—The filariated mosquitoes upon which he made his observations were not bred out and thus in confinement from the moment of their emergence from the pupa state; they were free mosquitoes obtained from a room where filariated persons slept. A few of the mosquitoes that were captured doubtless had imbibed blood weeks before and already contained advanced stages of the metamorphosis. They were imprisoned and never fed, consequently they died about the sixth or seventh day, when they were microscopically examined. Manson evidently believed that their last meal of blood was their first.

Manson has remarked¹ "that various stages of the metamorphosis were occasionally to be seen in the same mosquito." Such a thing never occurred to me, and is inexplicable except on the supposition that his mosquitoes had imbibed filariated blood on several different occasions.

In the following details my observations differ from those of others, who have worked at this subject.

(1) Pressure of the cover-glass more particularly and endosmosis are the cause of rupture and escape of material at the anus in the young filariæ; such is not a natural phenomenon; it will not happen if the thorax be teased out in Müller's fluid and examined without a cover-glass or with a small piece of cover-glass.

(2) After the meal of blood is digested, the mosquito's stomach and intestine contain no filariæ.

(3) The filariæ after imbedding themselves in the thoracic muscles lie quiescent until about the fourteenth or fifteenth day, when very slight movements can sometimes be detected.

(4) I have been unable to satisfy myself that the embryo filariæ cast their sheaths before leaving the mosquito's stomach; when seen in the thorax they appear to have lost the long collapsed sheath following tail; the sheath may however have only shrunk or it may be filled out by the worm, which has already grown longer and thicker; the tail is peculiar in the early stages, which may be due possibly to retention of the sheath.

(5) The filariæ, which emigrate to the thorax, do so directly they are withdrawn from the human host; those that are to be seen in the mosquito's stomach several hours later are they, that for some reason, whether being too young, or from injury or from having been already acted upon by the digestive juices, are not destined to enter upon a metamorphosis. Loss of sheath, striation of body, changes in the body protoplasm in them are due to endosmosis and digestion.

(6) No apparent sheath can be seen in the embryo filariæ in freshly drawn blood, but a flagellum-like body generally following the tail; sometimes the flagellum-like body is momentarily protruded from the head, and *pari passu* disappears from the tail; this only occurs when the worm is swimming tail first; such appearances cannot be seen in every embryo, and I am inclined to think that it is not normal. The flagellum-like body is the collapsed sheath, which can only be diagnosed as a sheath when endosmosis has taken place. Those who have figured the embryo, have represented a worm inside a distended sack; such appearance is unnatural. The purpose of the sheath is possibly to anchor the worm to the side of a blood vessel when the latter is resting.

Manson in his recent work,² p. 460, has remarked:—"It is also manifest that the purpose of the 'sheath' with which it is provided while circulating in the human host, is to muzzle the embryo filaria and prevent its breaking through the blood-vessels, and so missing its chance of gaining access to the mosquito."

If any should care to decide the question for himself, let

¹ *Australasian Medical Gazette*, March 20, 1899.

² *Ibid.*, June 20, 1898.

³ "Parasites of Man," by Rudolph Leuckart. Young J. Pentland, 1886.

⁴ *Trans. Linn. Soc. Lond.*, pp. 367-8, 1884.

¹ *Op. cit.*, p. 379.

² "Tropical Diseases," Cassell & Co., Ltd., 1898.

him prepare a slide of filarated blood and paint a little oil round the edge of the cover-glass to prevent evaporation and examine under the microscope twenty-four hours afterwards, when a certain amount of coagulation and crystallisation has taken place; this forms some resistance to the filariæ and they may be seen crossing from one edge of the cover-glass to the other in a tortuous but definite course with the collapsed sheath following tail.

I cannot agree with Manson that the sheath muzzles or impedes the filaria in any way; normally I believe the sheath is never separate from the body. The embryo in freshly drawn blood wriggles about but never seems to leave the same spot; this peculiarity was considered due to some impediment caused by the sheath, but the embryo of *Filaria immitis*, which is not possessed of a sheath, wriggles precisely in the same manner.

(7) Some writers would lead you to imagine that there is but a single pair of adult filariæ in each filarated subject; judging from analogy of what occurs in other animals harbouring filariæ, I believe that there are generally a good many present, a dozen or so, or possibly in some cases fifty. The number of embryos that are to be found in a drop of blood is some criterion of the number of adults in the subject; if the embryos are scarce, it is likely that there are few adult females, but if plentiful it is probable there are many females.

It is not known how long the embryo filaria lives in the blood, probably it is several months, and probably the adult worms live several years.

Provided a filarated subject could prevent reinfecting himself, it is very likely that in course of five years, he would be entirely free from the parasites. To accomplish this, it might be wise to emigrate to a county where there are no mosquitoes, and failing that, to sleep under perfect mosquito-net bed curtains.

Fortunately it is easy to rid the house of the *Culex ciliaris*; it appears that this insect was introduced into Australia;¹ it will not go wild but always frequents habitations, breeding in receptacles holding water in or about the house; such receptacles should be covered with gauze, net, perforated zinc or other material to exclude mosquitoes; cattle and poultry water troughs should be emptied out at least every ten days, as by so doing, the mosquito larvæ could never mature; it takes fourteen to twenty days from the mosquito egg to the perfect insect.

In this investigation the following methods were found the best. In breeding "house mosquitoes" it is necessary to obtain their eggs or larvæ. Galvanised iron washing tubs are convenient vessels wherein to rear the larvæ; these are filled with fresh water and placed in a shady spot; into them is put a handful of rotting leaves and a small piece of flesh, preferably flesh that has passed the putrefactive state in water, having been converted partly into adipocere. When animal matter forms part of the diet, the larvæ grow faster and to a larger size. The larvæ soon die should the water become foul. In a fortnight or so the larvæ will have changed into pupæ; by means of a miniature scoop-net (the size of a tablespoon) made of wire and mosquito net, the pupæ are transferred to a glass vessel of water such as a fish-bowl (about six inches in diameter at the mouth). The mouth of bowl is covered with muslin, the material known as "white lino," was found very serviceable; mosquito net is not suitable, as mosquitoes can, when they try, creep through the meshes, especially when the net is stretched tightly. The pupæ do not require food, and in a day or two the perfect insects will have emerged from them. The male mosquitoes are easily distinguished by their large feathery antennæ; they do not suck blood. Transference of mosquitoes to a glass cell is performed by means of a "collecting tube"; this is a hollow glass cylinder conveniently four inches long and one and a half inches in diameter, one end is covered with mosquito net, whilst a cork is loosely fitted to the

other; pieces of Argand gas-lamp chimney make good collecting tubes.

Glass cells, about ten inches high and six inches in diameter, are convenient wherein to store living mosquitoes; they are fitted up as follows:—At the bottom is placed a little dry sand, also a vessel holding three or four ounces of water; the sand serves to weight the cell and steady the water vessel; into the vessel of water is put two or three bits of straw or cork, this is to assist the mosquitoes rising from the water; as the mosquitoes age they get infirm and frequently get drowned unless they reach some floating object. Over the mouth of the cell is stretched a piece of wet lino and tied tightly with twine; when the lino is dry a circular hole an inch in diameter is cut out of the centre, and this hole is covered with a watch-glass, concave side upwards.

The transference of mosquitoes to a glass cell is done in the following way:—The mosquitoes are allowed to escape under the mosquito-net curtains; the cork being removed, the mouth of the "collecting tube" is placed over a mosquito, which then flies up the tube; the cork being now replaced the tube is brought close to the glass cell, the cork being directly over the watch-glass; the cork is removed and the tube put right on to the watch-glass, and at the same time the watch-glass is slid aside, the open mouths of the tube and cell are now together; a puff of air blown down the tube causes the mosquito to fly down into the cell; the watch-glass is again placed in position. By such means a dozen mosquitoes might be put into a cell in a minute without any danger of injuring them.

Female mosquitoes bred out by me were put into an empty cell of the capacity of forty ounces of water and sent to the home of the filarated subject, who liberated them under the bed-curtains upon retiring; next morning any with distended abdomen she captured by means of a collecting tube transferred back to the cell and returned the same to me; they were again liberated under curtains and transferred to larger vessels. In the cell storing mosquitoes a section of ripe banana is suspended; it was found best to cut the banana at right angles to its length in pieces one and a half inches in length, with the skin left on. Moulds very soon grow on the cut ends when the mosquitoes prefer to pierce the rind and thus get at the sound tissue. It is advisable to remove the piece of banana and replace by fresh every three or four days. Should the air in the cell become foul from the decomposition of banana, or from the odour of mould fungi or the water at times contaminated by banana juice, it is advisable to liberate the mosquitoes under a mosquito net curtain and transfer them to a clean cell. It is also well to place a plug of cotton wool in the hole in lino and over this the watch-glass, concave side down. The cells are placed in a room in a house where the light is subdued, or shaded by brown paper from too strong a light. Half a dozen mosquitoes is a sufficient number to put into one glass cell of the capacity of one hundred ounces of water.

When mosquitoes are required for examination, they are liberated under the curtains, captured and killed in the entomologist's cyanide bottle, or by means of chloroform, &c. Two pairs of ciliary forceps are useful with which to pull off the wings, legs and head; afterwards the body is divided by dissecting needles into thorax and abdomen, and each portion examined separately, teased out in water, or better in Müller's fluid with or without a cover-glass under a magnification of fifty diameters.

The following is a short account of the life-cycle of *Filaria Bancrofti*:—Commencing with the mature parasites in the human subject; these are three or four inches in length by $\frac{1}{16}$ " in breadth, they live in the lymphatic vessels; they produce the embryo filariæ, which are $\frac{1}{16}$ " \times $\frac{1}{32}$ "; these latter live in the blood-vessels, swimming about when the host is sleeping and resting themselves when he is awake.

Mosquitoes when biting a filarated subject during the night withdraw together with blood some of the embryo

¹ *Proc. Linn. Soc., N. S. Wales, Vol. iii. (Series 2nd), p. 1718.*

filariæ. Soon after the embryos reach the mosquito's stomach they pierce the stomach wall and find their way to some muscular mass, particularly the thoracic muscles, in which they imbed themselves. There, nourished by the mosquito's plasma, they grow at a prodigious rate, becoming longer and thicker, and assume by the fifth day the appearance in which a distinct line, the rudimentary intestine, can be seen from the mouth to the anus. The body protoplasm, at first homogeneous, has been changed into large cells with numerous vacuoles. In ten days the intestine presents a double line, the large cells have given place to very small cells; from this time on to the seventeenth day most remarkable changes occur, too intricate and difficult to describe. In seventeen days thereabout the young filaria has attained its maximum development as far as its life in the mosquito is concerned. It now awaits the chance of gaining entrance to the human host, in the event of which we presume that it will start upon a second metamorphosis, the final alternation of generations, in which it grows to the length of three or four inches and becomes sexually mature.

It remains to be proved that these young filariæ will become sexually mature in the human host. I have elsewhere¹ suggested how this might be accomplished, viz., by inducing a life-sentenced prisoner to swallow some mosquitoes bearing filariæ on condition that he be given a free pardon.

Besides proving that the *Culex ciliaris*, Linn., is an efficient host for *Filaria nocturna*, I have shown that two other species of mosquito are not hospitable, viz., *Culex notoscriptus*, Skuse, and *C. annulirostris*, Skuse. Both these mosquitoes will live in confinement at least twenty days. *Culex notoscriptus* sucks out plenty of embryos, but, as far as I have seen, none of these ever migrate to the thorax; they appear to have been killed by the salivary juice. Only rarely do some embryos migrate in the case of *Culex annulirostris*; after two days, however, any that did reach the thorax have died and been absorbed. Other mosquitoes have been experimented upon, but as I have been unable to keep these alive sufficiently long for the final stage of the metamorphosis, it is impossible to say definitely that they are not hospitable, yet everything tends to that conclusion.

In the case of *Culex hispidosus*, Skuse, and *C. vigilax*, Skuse, these two species live about seven days in confinement, and a number examined about that time contained no filariæ. In the case of *Culex nigrithorax*, Macquart., *C. procax*, Skuse, and *Anopheles musivus*, Skuse, I have been unable to keep them alive more than three days. A good many experiments were made with *Anopheles musivus*; this mosquito sucks out a large number of embryos, and the most of these migrate to the thorax.

For the scientific names of the mosquitoes I am indebted to Henry Tryon, Esq., Entomologist to the Queensland Government. Thanks are due also E. S., the filariated subject, without whose assistance this investigation could not have been carried out, and Manson's important discovery might for some time to come have remained unbelievable.

Deception Bay,
Queensland,
May, 1899.

Added June 1.

A number of mosquitoes imbibed filariated blood on April 26 and the final stage of the metamorphosis did not occur in them until May 31, i.e., thirty-five days. The weather was cold.

It has occurred to me that the young filariæ may gain entrance to the human host whilst mosquitoes bearing them are in the act of biting. The entrance of warm blood into the mosquito may excite the young filariæ, in consequence of which they pierce the œsophagus and pass down the proboscis into the human skin. In this way

injury from the human digestive agents would be avoided. It is not unreasonable to suppose that, like water, the digestive fluids would soon kill the young filariæ, but it is probable that those that may have been set free by rupture of the mosquito's body would immediately pierce the mucous membrane and enter a lymphatic or other vessel.

A MEDIEVAL PLAGUE REPORT.

In the *Wiener Klinische Rundschau*¹, No. 19, Dr. Senfelder gives an account of the oldest Austrian official plague report, that by Jakob Horst, dated 1588. He includes under plague or pestilence all epidemic diseases which are usually and rapidly fatal, and attack the "noble organs," head, heart, and liver. "There are many other epidemics which do not so greatly plague the noblest organs nor kill so rapidly nor so many, and these are not yet pestilence." He distinguishes two great divisions: *divinæ*, due to God's wrath, and to be treated less by earthly than by spiritual means; and *naturales*, caused by more moderate "wrath" and therefore curable. These may be further classed in three subdivisions: *perperacuti*, *peracuti* and *acuti*, according as death occurs in two to three, six to seven, or over seven days. Genuine pestilence includes not only "fever in which the blood inflames and ferments and throws out poisonous boils" but "every great sickness which rapidly destroys many." Such as (1) the English sweat "not improperly called *ephemera pestis* by some of the learned." (2) "Terrible putrefaction of the blood, so that limbs fall off and the patient loses memory," perhaps a reference to the plague of Athens described by Thucydides. (3) Fever with boils or buboes "the commonest in these days." (4) Fever with pocks, "of various kinds, but the black are the worst." (5) Burning fever with spots and petechiæ. (7) Toxic apoplexies and epilepsies which in times of pestilence become infective. Sometimes the plague is preceded by an incubation period of ten to twelve days with flying pains in the limbs, fainting, cold sweats, sudden collapse, altered expression, somnolence, exhaustion, &c., signs which require the immediate isolation of the patient. As internal *causæ antecedentes* he mentions accumulation of evil humours and their putrefaction, which call for purgative treatment. External causes are the air, especially near diseased persons, and "all things which catch and retain bad air, as skins, wool, silk, &c., also bad food or drink and fright."—*Janus*, June and July.

THE SIBERIAN RAILWAY AND PESTILENCE.

Next year will be devoted to opening up the Trans-Baikalian section of the Siberian railway traversing the district of Akcha, and the *Semaine Médicale* reminds us that this is the region in which two Russian physicians, MM. Beliansky and Rechnetkow, discovered a disease closely resembling the plague, a febrile disorder with painful buboes, nearly always fatal in two to four days. Buboes are sometimes absent, and the disease then assumes a pneumonic aspect. As the rat helps to spread the plague, so this disorder is carried by a small rodent called locally Sabagan (*Aretomis Bobæ*) and the disease is therefore called by the natives the Sabagan plague. The danger of the importation of this and other eastern diseases into Europe will obviously be much increased when the Yellow Sea and the Sea of Japan are brought within a fortnight's journey of Paris and London. Still more will this be the case when Central and South-east China, the native homes of plague and leprosy, are opened up. China will then assume a hygienic importance rivaling its present political one.—(*Revue Scientifique*, No. 22), *Janus*, July and August.

¹ Voir *Janus* iv. 5, p. 255. Nous avons cru ne pas devoir supprimer le compte-rendu ci-joint.—RED.

¹ *Australasian Medical Gazette*, March 20, 1899.

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THE

Journal of Tropical Medicine

NOVEMBER, 1899.

HYGIENE IN CAMPS.

In the campaign against the Boers several important health questions are sure to arise to which answers will be given in due time. One of these, which is already discussed in these columns, is the susceptibility to malaria under the conditions described. There is also the kind of wounds that the new weapons are likely to produce, and the treatment of the wounded soldier. This side of the subject will no doubt be carefully considered by the army medical men and the eminent civil surgeons who have been appointed as consultants. The Secretary of State for War is to be congratulated on the step which he has taken to secure for the British forces the best surgical skill available. There can be no doubt that it is a wise as well as a humane policy, that nothing should be left undone which science can suggest, to counteract the terrible injuries inflicted by modern weapons of war. We are already hearing of the destructive effect of the Melinite and Lyddite, the Mauser and the Lee-Metford; and the number of killed and wounded is gradually rising on both sides. There will be great need of the services of nurses and doctors, and we are glad to see that

the preparations have not been lacking in this respect.

Battles naturally lead us to think of the wounded, and there is little fear that all will be done to alleviate their sufferings. But campaigns are not made up wholly of battles, nor are sickness and death wholly the result of wounds. Experience, unfortunately, demonstrates that by far the greatest amount of disease and mortality in campaigns are due to causes unconnected with the battlefield. Never again is the disastrous mortality of the Crimea likely to be repeated. It is an object lesson of the past which will always be remembered, and which will, under every circumstance we hope, be avoided. More than this, however, is required in these days of sanitary science. There is a splendid field for the sanitary officer to save the army from preventable sickness and disease, and to so place at the disposal of the General a high percentage of the force.

With so strong an army service corps there should be no difficulty in the men receiving good substantial food and adapted to the hard work that they have to accomplish. We hope there will be no false economy in the rations as regards quantity, quality or variety. When hard work has to be done, under trying conditions in all weathers, the food supply and its proper cooking are very important elements in the maintenance of the health of the troops. It is not only when the soldier is sick and has to be nursed that attention should be paid to his food, but also when he is well, in order to keep him well and secure from him his fullest energies. We have placed the food supply before the water supply, for in these days so well known are the injurious results on the system of an impure or bad water supply that the provision of a good supply, or the prevention of contamination of the supply at hand, is not likely to be neglected. If it is neglected typhoid and dysentery will soon make themselves conspicuously prominent. The sanitary officers will always have to be prepared for contingencies. It is often as important for the enemy to cut off the supply of a town as to bombard its inhabitants. This appears to have been attempted

with more or less success in South Africa, and it is accordingly necessary always to be provided with an alternative supply which shall be sufficient in quantity. The hygiene of the camp becomes particularly important in the case of besieging or being besieged, and too much attention cannot be paid to the disposal of excreta and waste products as well as to the burial of the dead, for the more perfect the arrangements are for these, the less risk there is to the army.

We note that small-pox has broken out in the Cape. We hope that adequate precautions will be taken to prevent its getting among the troops. Further—though the chances may be remote—the fact should not be lost sight of that plague exists now, or existed a short time ago, in the vicinity of Delagoa Bay. It is necessary that this fact should not be forgotten, and that every precaution should be taken that this disease by no possibility should reach the scene of warfare. From this brief sketch it will be seen that there is something more to be done in the present campaign than healing of the soldiers' wounds, and we trust that the organisation for the prevention of disease is on an equally adequate scale.

BANCROFT ON FILARIA.

THE paper which we publish to-day on Filarial Metamorphosis, by Dr. Bancroft, is one of importance, not only as confirming in most particulars Manson's original investigations, but as probably correcting these in one important particular, and as showing that what is true of the filaria in China, in India and Egypt, is also true of the filaria in Australia. We would remark parenthetically that it is a singular circumstance, and one not very much to the credit of the tropical section of the profession, that Manson has had to wait just twenty years for adequate confirmation of his discovery. It is true that soon after the fact that the mosquito acted as intermediary host of the filaria was published, Sonsino and Lewis confirmed the observation in part; they failed, however, to follow out the final stages of the metamorphosis. Grassi made analogous observations on the metamorphosis of

filaria recondita in the dog flea; but neither did he nor, so far as we know, did any one else, until Bancroft took the matter up, add materially to what Manson had told us about the metamorphosis of filaria nocturna.

Apart from the confirmation it contains of what had been done already, a principal value of Bancroft's paper lies in the fact that he has rendered probable, if he has not quite proved, that the metamorphosis of the filaria in mosquito is not concluded in seven days, as Manson supposed, but that it requires at least sixteen days to complete, at all events in the species of mosquito, *Culex ciliaris*, the Australian observer experimented with. Further experiments are required, we think, before unqualified assent can be accorded to this; for it may be that species and atmospheric temperature have a powerful modifying influence in accelerating or retarding the process of evolution in the young filaria as they undoubtedly have in the malaria parasite and in proteosoma. It is curious to note how circumstances in Manson's experiments seemed to conspire to uphold the idea, then current among naturalists, that mosquitoes fed only once and died so soon as they had deposited their ova. It would seem that they feed twice or oftener after impregnation, and do not die, as a rule, before the third week; unimpregnated insects, and occasionally impregnated insects, much later. Ross last year taught us, what ought to have been a self-evident fact, that to live mosquitoes, like other animals, must feed. Bancroft has now given us a technique which simplifies the feeding of experimental insects, and which will render future experiments with these insects very easy.

It is not a little curious to note how Bancroft exposes himself to the suspicion of having fallen into the very error of which he has convicted, or probably convicted, Manson. While insisting on the necessity of feeding mosquitoes, he ignores the same necessity for filariæ. He says "the young filariæ died after three or four hours immersion in water," and from this he infers that drinking water is not the medium in which the filaria is transferred to the human host. It occurs to us to ask, if it may not be that the

death of the filaria in clean water is brought about by starvation? It has been experimentally proved that another miniature nematode — the embryo guinea-worm — lives longer in muddy water than in clean water, probably because it can get food in the former.

Bancroft's views on the nature of the filarial sheath, on filarial ecdysis, and on one or two minor points require modifying, we think; they are not in accordance with the findings of other competent and careful observers. The paper on the whole, however, is one of very great value; its appearance at the present juncture, when the many unsolved questions in connection with the relation of the mosquito to the germs of tropical disease are pressing for a solution, is most opportune. It indicates directions for new investigations; amongst others, failing opportunity for the experimental filariation of the human subject, corresponding experiments on the lower animals, say of the dog, with filaria immitis or filaria recondita, much information bearing on human filariasis might be acquired in this way. Bancroft's suggestions that the filaria may be acquired by swallowing a filariated mosquito, or by the bite of such an insect, seem to us to be highly improbable, and hardly in conformity with many known facts in regard to the relationship of drinking water to filarial disease, or with the comparative freedom of Europeans from filarial infection even in highly filarial countries.

Recent malarial work and the interest in filariasis that is sure to be revived by Bancroft's paper, make it extremely desirable that the English observer be provided with a good work on the mosquito. Such a work should deal in a practical way with the anatomy and habits of this order of insect, with the classification, description and illustration of the various known genera and species; with the technique of their dissection, and with the methods for experiments in malaria and filariasis. Such a work would find a ready sale.

THE CAMPAIGN IN SOUTH AFRICA:

I.—CLIMATE AND DISEASE.

THE campaign at present proceeding in South Africa is being conducted in Cape Colony, Natal, the Transvaal and the Orange Free State. The actual theatre of the fight is for the moment in Northern Natal, but the line of communications run to Cape Town, and the future seat of activity will be in the Transvaal itself. From Cape Town to the Limpopo River bounding the Transvaal on the north is a distance of 1,200 miles, and from east to west the troops are scattered over a distance of between 400 and 500 miles. In so wide a range of country well-nigh every variety of climate is to be met with; and an analysis of the districts in which actual campaigning is to take place, may be of service at the present juncture.

Natal: Its Position and Climate.

The Colony of Natal borders on the Indian Ocean and extend between 31° 14' and 27° 20' south latitude. It is a sub-tropical country therefore, but like all countries south of the equator, the climate is much less "tropical" than is the case in countries in a corresponding latitude north of the equator. In fact, Natal has come to be considered a sanatorium, more especially for persons suffering from pulmonary complaints. The mean temperature of the Colony throughout the year is almost 65° F. Lying as it does just outside the tropical zone, it is of course hot, but the heat in summer is much modified by cool winds, rain and thunderstorms. Summer begins in October and ends in March, and winter lasts from April to September.

In summer the principal rain-fall occurs. A hot north-west wind from off the equatorial plains of the interior of Africa occasionally prevails in the north, causing intense discomfort, and at intervals during the summer, hail and thunderstorms are apt to cause a good deal of destruction to growing crops. During the winter (April to September) slight frosts occur in the higher ground. The Drakenberg range of mountains runs along the western portion of the country some 200 miles from the sea coast, the range presents cliff-like peaks, attaining in some instances a height of 10,000 feet; and from the range ramifications of hills slope eastward. Travelling inwards from the coast, the country presents a succession of terraces or plateaus, rising to a height of over 4,000 feet, until the Drakenberg range is reached.

The average temperature of the Colony taken in towns such as Durban, Pietermaritzburg, &c., is as follows:—

	Highest	Lowest	Mean
Dec. to Feb. (Summer)	97.5	53.3	71.2
June to August (Winter)	83.4	31.9	56.7

Average Rainfall 33.50 inches.

Fires are not necessary even in winter, until a distance of some 50 miles from the sea coast is reached.

The diseases of Natal are not of that virulence that we are wont to associate with countries lying so near the tropics. Malaria is not the scourge it is further north. Black-water fever is rare, bowel troubles are not frequent, in fact, epidemics are the exception.

Transvaal.

The Tropic of Capricorn passes through the northern part of the Transvaal, so that the main part of the Transvaal is subtropical. The limits are 22° 15' long. and 28° south latitude. In mileage the country measures 500 miles in its greatest length, and about 400 miles in breadth.

The configuration of the Transvaal has been aptly compared with a saucer, the lip of the saucer representing the mountains encircling the more hollow land in the interior. The hollow is, however, merely relative, as the whole country is an elevated plateau, some 3,000 feet above sea level. The lofty Drakenberg range extends northwards from Natal into the Transvaal, gradually breaking up into offshoots, which broaden out until they are but little above the surrounding plateau.

Although lying partly within the tropics, the Transvaal plateau is so high above the sea level that the country enjoys for the most part a salubrious and invigorating climate to the east, where the Drakenberg highest spurs range. The climate is moist and misty, and the rainfall some sixty inches. As the western portion is traversed, however, the air is much drier, and the rainfall diminishes in amount from sixty in the extreme east to thirty in the central area, and some twelve inches only on the western border. The dryness in the west is caused by the absence of high mountains and the proximity of the Kalahari Desert. It is for this reason that the Western Transvaal is so sought after by invalids.

The only parts of the country where malarial fevers of the African type exist are in the extreme north, along the banks of the Limpopo, or Crocodile river, and again in the country of the Swazis. With these exceptions, however, the country is healthy and favourable for European constitutions.

The Orange Free State lies wholly to the south of the Transvaal, reaching from 31° to 27° south latitude. The whole country consists of a lofty plateau, having an average elevation of some 4,500 feet above sea level, and presents a series of wide undulating plains with rocky hills interspersed. The climate is dry and salubrious, and the atmosphere clear and invigorating.

Such are the countries in which our troops are engaged in their campaign against the Boers, and it is seldom that British troops are called upon to fight in so favourable a climate, or where the dangers from the great enemy, malaria, are so remote.

The Troops Engaged.

The soldiers employed in the campaign may be arranged in two groups. The first group, those proceeding direct from Britain, consists for the most part of men who have never been exposed to malarial influences, who have never been out of Britain. Contrary to what usually is the case in our campaigns, these men will stand a better chance of remaining fit than those who have been drawn from Indian garrisons. They are not likely to contract malarial fever in Natal to any great extent, and being free of infection may be presumed to be able to endure the exigencies of war in so healthy a climate. Amongst the men from Britain are a number of Reserve men, many of whom have no doubt served in the tropics,

and have been exposed to malaria. Seeing however that they have resided for a shorter or longer period at home, it might be hoped that their blood would be free of the malarial organism. This, however, is a pure assumption which may not stand the light of experience, and amongst the troops which have reached Natal from Britain, it may be found that those of them who are old Indian soldiers are more liable to malarial attacks than their home-reared *confrères*.

The Indian contingent, some 5,000 in number, are on a different footing. They come already charged with malaria to a country where endemic malaria is rare. An upland country, where the clear invigorating air thwarts the outbreak of, and actually serves to eliminate, the poison. But dwelling in a comfortable home and campaigning in the veldt are two totally different things. The nights in Natal and especially in the Transvaal and Orange Free State are cold actually, and also relatively to the heat of the day. Where marked divergence of the kind takes place the bivouac is attended by marked danger. On the battle fields of the Peninsula, on the more elevated tablelands of Central Spain especially, although malaria claimed its victims, tetanus was more dreaded, more fatal and more prevalent. In the Soudan the early morning chill is the greatest enemy to health, and wherever a dry atmosphere prevails, be it the Soudan, the Orange Free State, Rajaputana, or elsewhere, the early morning cold has to be guarded against and provided for.

Natal, therefore, although malaria can scarcely be said to be endemic there, although the elevation of 3,000 to 4,000 feet is especially favourable to restoring health to persons suffering from chronic malaria, yet bivouacing on the veldt, hungry, fatigued, wounded it may be, and drenched to the skin are eminently provocative of latent malaria developing. Troops from India, therefore, may expect to suffer from malaria in a greater proportion than they did in India whilst resident in barracks. Already (Oct. 30) we have news of that very condition occurring. The Colonel of the Gordon Highlanders had his arm wounded by a bullet, the next news we receive, a few days later, is that he is suffering from ague. This is the story of one of the first men from the contingent of troops from India we have received, and it is to be feared the condition will be frequently repeated.

Another point to be determined is, will malaria attack the troops from Britain who have never been in the tropics before? If so, then one of two things must obtain—either that malaria was contracted from the environment in Natal, or from the malarial-infected men from India. The first alternative implies that malaria is endemic in Natal. This we know is true to a limited extent only, therefore any marked outbreak implies inoculation from infected neighbours. But inoculation means the mosquito—a special variety of mosquito—and we have no authority as yet to say that the malarial-transmitting mosquito exists in Natal.

Attention to these queries will add considerably to our knowledge of malaria generally, and to malaria in Natal in particular. They may be summed up as follows:—



The Illustration represents the Thorax of an Adult, aged 26 (Sergeant of Infantry), with a 5^m/₁₆ bullet embedded in the Lung.

The Radiogram was taken with an *Electrolytic Break*, double-coated *Schleussner Plates*, *Intensifying Screens*, and *Voltohm Apparatus*. Exposure: a fraction of a second.
Photograph by the Voltohm Company. Lent by Isenthal, Potzler & Co. *Beile & Danickson, Ltd., London.*

(1) Is there a marked difference in the health of the soldiers from India and Britain respectively as regards malaria?

(2) Do the soldiers forming the corps proceeding direct from Britain suffer from malaria; if so, to what extent? Is it confined to soldiers who have been in the tropics before, or are men who have never been out of Britain until now attacked? J. C.

II.—NOTES ON FIELD SURGERY.

By WM. DICK, M.B., F.R.C.S. Edin., Major R.A.M.C.

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Two factors in modern warfare will greatly modify the practice of surgery in the field, and they will probably both be evident in the present South African military operations; they are, first, the small calibre rifles with which both sides are armed, and second, the thorough application of aseptic or antiseptic methods to gun-shot injuries. At present we are not in possession of much information as to the effect of Mauser and Lee-Metford bullets in living human beings. It is expected that these bullets, when only passing through the soft structures, will make comparatively small wounds which will readily heal under modern surgical treatment. The case, however, will be quite different where bony structures are involved. Experiments go to prove that when the shafts of long bones are hit, and, in fact, any compact bone tissue, that there will be extensive comminution and fissuring.

Cancellous tissue, on the other hand, will probably have wounds more of the character of those in the soft tissues of the body. The antiseptic methods with voluminous dry absorbent dressings will greatly tend to the practice of conservative procedure, so that proportionately there will be fewer amputations than in previous wars.

The following notes are based upon a certain amount of experience in the field, and also on a considerable amount of experience in the later treatment of wounds which have been received in the recent Indian frontier and later Egyptian campaigns.

To commence with, I think that Senn's dictum that no touching of wounds is to be done by those who first pick up the wounded, cannot be too strongly insisted on. The sole duty of the stretcher-bearers will be to apply at once the first field dressing so as to close up at once wounds, and to apply splints to immobilise fractures. The only exception to this rule is where continuous hæmorrhage has to be at once stopped at all risks of infection. It is quite impossible for either surgeons or bearers on the actual field to render their hands and instruments aseptic, therefore the less touching and examining of wounds the better.

When a wounded man is found on the field, it will be comparatively easy without touching the wound to ascertain whether hæmorrhage is going on, or whether there is a fracture. If neither of these conditions exist, the first field dressing will be firmly applied, and the man is then ready to be removed

to the collecting and dressing stations. If there is fracture without hæmorrhage the part must be made immobile by splints made of the man's accoutrements and clothing, always remembering that natural splints, viz., the sound limb in the lower extremity and the body in the upper extremity are to our hand to aid us in rendering a fracture safe for removal from the field. If there is manifest hæmorrhage this must be controlled and at once, and I think the only way to do this is for the bearer to compress the bleeding points with his fingers and thumbs until a compress can be applied locally, and a tourniquet can be got ready, improvised from the man's braces, belt, or strips of his clothing. To exemplify how time and perhaps life is lost when methodical treatment is not applied, the following experience occurred to myself when examining a class for ambulance certificates. Three patients were put out labelled as having the following injuries:—(1) Hæmorrhage, wound of radial artery; (2) compound fracture of leg; (3) compound fracture of upper arm. A stretcher detachment was sent to search for wounded, and found all three. They promptly put splints on the man with the broken leg, and carried him into the collecting station on the stretcher; they then went back and did the same by the man with the broken arm; lastly they brought in the man with the hæmorrhage, having put on a compress on the wound and a tourniquet on the brachial. Needless to say that what they ought to have done was to have arrested the hæmorrhage first, put the man with the broken leg on the stretcher, having put splints on it and on the broken arm, to have brought all three in together, No. 2 being carried, No. 1 and No. 3 walking with the help of the bearers. In wounds of the head, chest and abdomen of all kinds, nothing can be done except to apply the first field dressing. This sums up all that can be done for a wounded man on the field.

The next step is when the wounded are brought to the dressing station. Here a supply of dressings can be obtained, instruments, and boiling water to sterilise them; anæsthetics, and surgeons to administer them; assistants, and simple food and stimulants. Here the work is got through much more expeditiously where division of labour is systematically carried out amongst the surgeons and their assistants. The transport for the wounded to the nearest field hospital is here ready, and the wounded should be passed on as quickly as possible. The simpler variety of wounds can be sent on as quickly as possible without any interference here, as the first field dressing, if it has been properly applied, is quite sufficient. Wounds of the skull, chest and abdomen, I think also, after having been looked to and properly dressed, ought also to be sent on without any interference except what can be done quickly, such as the removal of a bullet that can be seen, or a loose piece of bone pressing on the brain. All compound fractures must have the wounds cleaned, dressed antiseptically, and splints re-applied, so as to put the broken bones and keep them during transport, in the most satisfactory position obtainable. The only operations, as a general rule, that can be done here are amputations in limbs that are thoroughly smashed, or merely hanging to the body

by shreds, and in which it would be a manifest danger to life to transport the patient with them, and the ligature of arteries in the cases of hæmorrhage, preferably by enlarging the wound and ligaturing above and below the injury in the vessel.

I do not quite see how, with any hope of success, an operation can be undertaken here for internal injury, even though some hæmorrhage may be going on, for the following reasons: These operations demand great skill, the possession of numerous appliances, and they take up a great deal of time which might be more profitably given to others. For these very reasons, however, the sooner they are sent off to the field hospital the better.

Having reached the field hospital, the treatment of the wounded does not differ in its essential aspects from treatment in any other hospital, civil or military, with the one great exception, that in all probability they must be moved to hospitals towards the base long before one would think of any movement after operation in stationary hospitals, but military exigencies demand, as a rule, that the hospitals in the front must be cleared as soon as possible.

(1) *Gunshot wounds through the soft parts* of the limbs will always be treated conservatively; of course vessels may require to be ligatured, nerves and tendons to be sutured, but the question of amputation will only crop up when symptoms such as gangrene, &c., supervene.

(2) *Gunshot wounds of the upper extremity*, with wounds of *joints* or *fracture* of bones will also, I think, be treated conservatively, except in the case in which there is extensive comminution with wound of the main vessel. In this case the only resort is amputation.

Here the subject of anæsthetics in the field will be dealt with in a few words. I think that *chloroform* is the only anæsthetic because, firstly, it is not so volatile as ether; secondly, anæsthesia can be obtained with a smaller expenditure; thirdly, it can be given without any apparatus. As regards the first two of these conditions, when it is considered that all supplies probably have to be sent long distances, it is evident that this is important in the field; as regards the third condition, all apparatuses are apt to get out of order, and the materials of which they are made do not keep in hot climates. As regards the safety of chloroform, the more I see of it the more convinced I am becoming that dangerous symptoms mean overdose, and the only way to avoid overdose is unflagging attention on the part of the anæsthetist. The chloroform should always be sent in bottles of not more than two ounces, stoppered, and sealed round the stopper with plaster of Paris. A cork with a dropper through it ought to be attached to the bottle which it will fit (the size of the bottle with the dropper cork will save a large amount that would otherwise be wasted if larger bottles had to be opened). It can easily then be given on a piece of lint.

To return to gunshot wounds of the upper extremity. Now that we are prepared to carry out antiseptic treatment as thoroughly as in any large hospital at home, and as the small bullets probably carry into the wound no clothing or anything else likely to give rise to sepsis, conservative surgery is the only logical method

to follow. No doubt we may get stiff joints and unsightly arms, but if position is attended to, as a rule useful limbs will be obtained. If the joints should become ankylosed, then the question of resection would come on at a later date, and under more favourable conditions. Should, however, the wound become septic, then a totally different condition arises, and amputation may be necessary to save the patient from exhaustion from continued suppuration.

(3) *Gunshot wounds with fracture, or wounds of joints in the lower extremity*.—The same conditions hold as in the upper extremity. The aim of the surgeon is to conserve the limb, and in order to do this he must practise the strictest antiseptic precautions, and keep his wounds free from suppuration. Of course in these injuries the question of amputation would arise, if in addition to the fracture the femoral vessels were injured. I don't think that the question of excision of the knee joint arises, as the best result one can obtain is a stiff joint, and this as a rule can be equally obtained by conservative treatment.

Of course in these injuries of the lower limb I think that the military necessity of emptying the hospitals at the front will weigh much more with the surgeon when determining the question of amputation, and that his judgment will lead him to amputate more often than he would in the upper extremity.

In the Soudan campaign of 1885, I found that those amputations which had been done by skin flaps more often required secondary amputation than those in which the flaps were made to contain all the tissues, and I called attention to this in the report of the Army Medical Department. Accordingly, I should continue to amputate always by skin and muscle flaps, unless I saw cause to alter my opinion. I think that in these flaps the vascular supply is better than in those with skin flaps alone, and this is important in view of early and jolting transport to the base.

One effect of the new small bullets will be that lodgment will not be so frequent as with the old bullets, therefore operations for extraction will not be so frequent. We have also now the X-rays to enable us to detect the presence of foreign bodies, and along with the apparatus supplied to troops in the field there is the excellent McKenzie Davidson localiser, which shows us the actual position of the bullet and its relations. This will abolish all searching and exploration in the dark, and will also tell us the best means of removal, whether by the track of entrance, or (which will frequently be the case) by making a new wound over the position where it lies nearest to the surface. A large number of these apparatuses in competent hands have been sent with our troops to South Africa.

(4) *Wounds of the head*, where the skull is not fractured, only require thorough antisepticism in their treatment; where there is fracture, without penetration, the symptoms will guide us to the operation of trephining. Where there is penetration with through-and-through perforation, i.e., without lodgment, again thorough antisepticism in our dressings and asepticism in our procedures are required, but here loose speculæ of bone ought to be removed, and depressed pieces must be elevated, and the trephine

will be required at all events at the wound of entrance for this purpose.

Where there is penetration without perforation, *i.e.*, with lodgment, the surgeon must decide for himself how much search can be made for the bullet or part of the bullet, but it cannot be very thorough. As regards the fracture itself, he must follow the same lines as in the previous case. After the bullet has been localised, the question of its removal will depend upon its position. In all cases the head must be shaved, and I think a gauze drain inserted a certain way into each wound to prevent accumulation, and the whole enveloped in a large absorbent dressing, is probably the best practice. Compression may come on shortly after the wound due to hæmorrhage. If the wounded vessel cannot be seen at either of the wounds so as to be secured, very little can be done surgically. Ice bags externally and a calomel purge is all that can be done. Compression later, due to the formation of an abscess, is different; it can very often be localised by its symptoms; if so, trephining ought to be done, an aspirating needle put in, and if pus is discovered an incision into the abscess made. The mortality in all these cases is sure to be large.

(5) *Wounds of the chest.*—If of the parietes only, ordinary treatment is all that is required; when the bullet is localised it can be removed. Cases of penetration in which the large vessels at the root of the lung have been injured will not be seen, as death will rapidly take place from hæmorrhage. In others, where the bullet has either passed through or lodged, no probing or exploration is permissible, and all that can be done is to render the wounds aseptic, and control the movements of the chest as far as possible. In these cases a considerable number of recoveries will be recorded.

(6) *Gunshot wounds of the abdomen.*—My remarks on these wounds are principally taken from Colonel Stevenson's paper read at the British Medical Association in August, and published in the *Journal* of October 21, 1899. In contusions and non-penetrating wounds, the treatment is simple, but the surgeon must in these cases be always on the look out for symptoms indicative of internal injury, *viz.*, the shock which nearly always attends injury to abdominal organs, and the blanching and perhaps dulness in the flanks which indicates intra-abdominal hæmorrhage; when these symptoms appear the same treatment must be carried out as in penetrating wounds.

In penetrating wounds nearly always operative interference will be required. Pain, nausea or vomiting, shock and blanching, will usually be present, and from these the surgeon must make up his mind as to whether there is injury to the contents of the cavity or not. If these are not recovered from in a few hours, and especially if they tend to increase, laparotomy, preferably by central linear incision, must be at once performed. Subcutaneous injections of ether should be given as a stimulant. Senn's hydrogen apparatus to confirm diagnosis is not likely to be at hand. If the operation is delayed until the hæmorrhage is excessive or until peritonitis has set in, it will probably be of little avail. No opium should be given, as it masks symptoms. Probably no extra danger is incurred by the operation, even in those cases in which

no injury to solid or hollow viscera is found. The most strict antiseptic precautions, with which all surgeons are familiar, must be employed. The central linear incision must be sufficiently large to allow of thorough inspection of the whole cavity. Sources of hæmorrhage must be first of all looked for and secured, then the whole bowel must be inspected, as also the solid organs; this must be done as rapidly as possible consistent with thoroughness. In gunshot injuries there will often be more apertures in the bowel than one.

If the apertures are far apart they should be sutured by Lambert suture transversely to the long axis of the bowel. Should there be considerable loss of tissue so that great diminution of the lumen would result, or should the mesenteric margin be injured, then excision of a portion of the bowel must be performed, and where several perforations are close together, one portion of the bowel can very often be excised so as to include them all. The enterorrhaphy will be performed according to the method best known to the operating surgeon. The vast majority of wounds will only require the Lambert suture. The peritoneal cavity will require thorough irrigation with hot boracic solution, and must be drained. For the first two days no food is to be given; for the next five, only fluid food. If the liver is wounded hæmorrhage must be arrested by packing with gauze, bringing the end out of the abdominal wound.

Wounds of the spleen will probably require splenectomy. Wounds of the kidney can be packed, the peritoneum sutured and an incision made in the lumbar region for the removal of the packing; in some cases nephrectomy will be required.

Wounds of the bladder require the wound in the viscus to be sutured, the peritoneum also to be sutured, and drainage to be maintained either by the urethra or probably by perineal section.

During operations the patient must be kept warm, and if there is great collapse from loss of blood, good might be obtained by the injection into the subcutaneous areolar tissue of sterile salt solution, half per cent., which is very readily absorbed and easily done. Too minute a search for bullets lodged in the abdominal cavity should not be made, as at a later period when they have been localised a decision as to their removal can be arrived at; of course, if easily found they can be removed at once. One must remember that the hospitals at the front must be emptied as soon as possible, and this fact militates against the success of all laparotomy operations; still I think they give a greater chance of recovery than leaving abdominal wounds to nature, as was the former common practice.

If there is a large number of wounded and the number of surgeons limited, the most attention will be given to those who are most likely to speedily repay the surgeons' efforts, and it would not be sound practice to spend a great deal of time on an abdominal case when a large number of less serious cases are awaiting treatment; consequently the judgment of the medical officers must necessarily be severely tried. Another point which contrasts with civil practice is that, whatever precautions the surgeon has taken against contamination in his operations, all sorts of

accidents not to the advantage of the patient may take place as he is passed from hand to hand and hospital to hospital along an extended line of communications until he reaches the base. The greatest economy has also to be practised in the use of surgical materials, as, however well stocked the hospital may have been, it is not always possible to foretell the amount required, nor is it always easy to get supplies replenished at the exact time when needed, as transport may break down or be captured by the enemy. Again, a large number of wounded of the enemy may require treatment, for whom it is impossible to make previous calculation.

III.—POSSIBLE CAUSES OF SICKNESS AMONGST THE BRITISH TROOPS IN SOUTH AFRICA.

By L. W. SAMBON, M.D. (NAPLES).

Lecturer to the School of Tropical Medicine.

THE Boers are a formidable enemy. Born and reared on the great rolling Veldt, they have become a fine, hardy race; they are splendid horsemen and wonderful marksmen; they are stubborn, dauntless, persevering. In the present war they appear to be well organised and powerfully armed with the latest and deadliest weapons. It is obvious, therefore, that the British Army, in its struggle for supremacy, will have to suffer very heavily from their fire.

The nature of the wounds inflicted by modern projectiles is so fully and ably discussed by Professor Dick that I need not enter into this subject. I believe, however, that it is only right to state that on account of the large bodies of troops employed and of the number of casualties which are likely to occur, the *personnel* of the Royal Army Medical Corps is numerically inadequate to meet the requirements of the campaign.

It is certainly gratifying to hear that such men as Sir William MacCormac and Mr. Frederick Treves have taken up the duties of consulting surgeons to the Field Force and that the Red Cross Society will provide men and equipments for the lines of communication, but the most essential requirement is a sufficient number of skilled surgeons at the front, thoroughly trained in the methods of modern surgery. It is our duty to see that the best possible aid be insured to those who are so generously risking their lives for the honour and power of their country.

It is probable that many more men will be killed outright on the field, because skull wounds and severe hæmorrhages will be far more frequent with the new projectiles; but, on the other hand, we may confidently expect a greater number of recoveries amongst the wounded, because these missiles produce more clearly cut and less septic wounds, and because the use of the radiograph will avoid those manipulations for the research of bullets and fragments which formerly were a great cause of sepsis.

The surgeon on the field whose dressing material may not be above suspicion should singe it before applying it to the wound. I have found this procedure very useful in cases of emergency. The use

of burnt cloth in the dressing of wounds is exceedingly old. The Japanese, in their last war with China, employed with great advantage carbonised straw.

Besides the casualties which may occur in the fighting line, we must not forget that sickness may break out amongst the troops and greatly hamper their movements.

South Africa is undoubtedly one of the healthiest countries in the world, but nevertheless it claims one or two diseases which, under favourable conditions, might cause far more havoc than the Boers' fire.

Previous campaigns in South Africa have taught us that we should be on our guard against malarial fevers, typhoid and dysentery.

Malarial Fevers are of very secondary importance in the pathology of South Africa, so far as it at present affects Europeans at their various stations, but we must not forget that they are exceedingly rife at certain low-lying, swampy districts, and especially along the sea-coast of Zululand. If the troops were obliged to occupy such districts for any length of time, malarial fevers would certainly prevail amongst them and prove as severe as those of any other malarious region.

In the Galeaka-Gaika campaign of 1877-8, and in the Zulu war of 1879, the British troops appear to have suffered principally from a mixed infection of enteric and intermittent fever, which gave rise to a certain amount of discussion as to the nature of the sickness. The term "typho-malarial fever" was applied to this mixed infection by Dr. Woodward, while serving in the Federal Army of the Potomac in 1861. This appellation may stand on the condition that it be clearly understood that it means a complication of two distinct infections, and by no means a specific disease or a peculiar form of either typhoid or malaria.

In a malarious region intermittent fever may develop in a patient suffering from typhoid, but far more frequently it is the typhoid fever which attacks a man actually suffering from malarial paroxysms, or which evokes into activity a latent malarial infection, just as it may give rise to an outburst of tubercular infection in another patient who may harbour Koch's bacillus. We know that the parasites of malaria may remain latent in the body for months, and even years, and, therefore, before admitting a local infection, we should ascertain whether the patient had been previously exposed to infection in other countries known to be malarious.

It is important to remember that in a typhoid patient, particularly during defervescence and convalescence, we may observe intermittent fever with chills. These symptoms have no connection whatever with malaria, but are due to other secondary infections, many of which are as yet unknown.

Our knowledge of malarial diseases has made such wonderful progress of late years, especially owing to the works of the Italian school, and to the recent important discoveries of Manson, Ross and Grassi, that we are now in a position to formulate judicious and appropriate prophylactic rules.

We know that in malarious countries all low-lying, swampy districts are the seats of infection, and that the fevers, in most places, show a decided seasonal

prevalence. In South Africa, as in Italy, they prevail in spring and in autumn. Since Laveran's discovery in 1885, we know that malarial fevers are due to special parasites belonging to the class of protozoa, and Ross's important experiments have proved that they are liberated from the body of one man and introduced into the body of another by the blood-sucking females of certain species of mosquitoes of the genus *anopheles*, which subserve them as intermediate hosts.

(siriasis) need not be feared, because it is unknown in South Africa. When military exigences compel the camp to be located in a malarious region, damp fires should be lighted in the evening to envelop the camp in a thick cloud of smoke. Mosquito nets should be used, or the men should smoke in their tents. Quinine may also be used as a prophylactic while residing in malarious districts and should be continued for several days after leaving the same. The dose should be from three to five grains daily.



THE "TORTOISE" TENT.

This tent, as supplied by the Military Equipment Stores, is suitable as a Field Hospital; it is portable, packs in small compass, and can be carried in sections by men or animals. The "Tortoise" tents have been adopted as Field Hospitals in the German, French, and other armies.

The life history of the principal malarial parasites is now pretty well known, and is most interesting in its various phases and details. Indeed, there are still many physicians who, having made no special study of parasitology, look upon it as romantic and incredible, while it is strikingly analogous to that of hundreds of other parasitic animals.

The troops should avoid as far as possible swampy districts, and when obliged to cross them, they should do so in the fulness of the day and through open ground, because mosquitoes usually come out at night and remain amongst bushes during the hottest part of the day. Thick gloves and gauze coverings for the head should be used, or kerosine oil might be smeared on hands and face. So-called sunstroke

The examination of the blood for malarial parasites should be insisted upon. The want of a microscope, or the inability to use it, are inconsistent with the practice of modern science. The demonstration of malarial parasites will render diagnosis certain, and will greatly help in the treatment of cases. It will certainly prevent the exceedingly injurious practice of cinchonising patients suffering from typhoid or other fevers, on the suspicion of malaria.

All pools near the camp in which mosquitoes are likely to breed should be drained or treated with kerosine oil, and this should be done likewise wherever the throwing up of earth for military purposes has disturbed soil drainage and given rise to pools and puddles.

Patients suffering from active paroxysms should be protected from the bite of mosquitoes, to restrict as far as possible the dissemination of the disease. The experience of Mauritius, and other places which at one time were free from malarial fevers, proves that a malarial patient may become a focus of infection, granted the presence of a suitable species of mosquito and other favourable conditions.

Typhoid fever is the most prevalent and most fatal disease in South Africa. In the Galeaka-Gaika war it was stated by the Principal Medical Officer to have been "undoubtedly the most serious disease during the war."

In the Zulu War of 1878, typhoid appeared at the head-quarters at Helpmakaar, and at Rorke's Drift, in the middle of February, accompanied by diarrhoea and dysentery. Helpmakaar became so unhealthy that it had to be evacuated. The troops were moved to Utrecht and Dundee, but the fever immediately broke out at both these places.

Typhoid fever is the most awful pestilence of warfare, not only from the number of cases, but on account of their severity and prolonged duration. Our knowledge of this disease is far from being perfect. Eberth, in 1880, described a bacillus which was subsequently studied by Koch, Gaffky, Sanarelli and others, and is now recognised to be the specific agent of the disease.

Typhoid fever is not an intestinal disease, as was at one time believed from the gravity which the intestinal lesions may assume during its course. Bacteriology has taught us that the typhoid infection is a general infection. The *bacillus typhosus* is found chiefly in the spleen, in the mesenteric glands, in the kidneys; its distribution in the body proves that it must enter the general circulation, and, in fact, it may be frequently demonstrated in the blood taken from the finger tip. It is rarely found in the stools before

the seventh or ninth day of the disease, and at an earlier date it may be found in the urine and sometimes in the sputum. It is evident, therefore, that its appearance in the stools is in consequence of elimination; this elimination may take place through the biliary ducts, the gall bladder may contain large numbers of typhoid bacilli.

It is an almost unanimous opinion that all typhoid

outbreaks may be traced to the polluting of the water-supply by the excrements of a typhoid patient. I do not contend in the least that water may not be a vehicle, and possibly the principal vehicle of typhoid infection, but I think it is very obnoxious to generalise a theory, however plausible it may be. Indeed, there are many outbreaks that cannot receive so comfortable an explanation. Some of them are strikingly limited, and their limitation is rarely in accordance with the distribution of the water supply supposed to be polluted.

A fact, which I think has not received sufficient attention, is that in a number of outbreaks the infection has been clearly traced to the dairy. Unfortunately, the prevailing water theory found an easy explanation of this fact by assuming that the milk had been diluted with polluted



THE "TORTOISE" TENT PACKED FOR CARRIAGE ON HORSEBACK.

water. In almost every case one or more typhoid patients were found at the farm which supplied the specifically infected milk. Their stools had been carelessly emptied into a dungheap and had thus percolated to the water supply.

I believe that typhoid contaminated milk has a far more important bearing than is generally supposed. It seems to me that it points very strongly to cattle as a possible factor in typhoid outbreaks.

Several other facts can be mentioned in support of this theory. Some outbreaks in Switzerland have been traced to the consumption of meat from diseased calves. Epidemics of typhoid fever are very frequent

in rural districts and at isolated farmhouses. These outbreaks in most cases cannot be explained by human importations.

Dr. James Allen, of Pietermaritzburg, from his observations in South Africa, came to the conclusion that typhoid fever depended chiefly upon infected cattle. He describes a specific enteritis occurring in calves, subject to relapses, and very contagious, and holds that the excrements of animals affected with this distemper, on gaining access in any way into the human body, will give rise to typhoid fever. He holds that typhoid fever thus arose in a great measure amongst the British troops in the Zulu war.

Typhoid fever is said to prevail also amongst horses. Servoles demonstrated the characteristic lesions in Peyer's patches. In 1881 the Paris Omnibus Company lost 1,500 horses from this cause.

In recent years we have been led to acknowledge more and more the important rôle that the animals around us play in the breeding and transmission of disease. From analogy with other diseases, all of which have a more or less wide zoological distribution, and especially from the facts mentioned above, I am inclined to look upon cattle and horses as possible factors in typhoid epidemics. At any rate, I think this possibility should not be overlooked in the adoption of preventive measures in the field.

A very important vehicle of typhoid infection is the common fly. Its importance cannot be overestimated. Water, milk, vegetables and meat can be rendered harmless by thorough cooking, but the flies which usually infest camps in enormous numbers may render all precautions useless. After having gorged themselves on the dejections of the patients they will come and disgorge themselves on the food already cooked, and on the drinking utensils.

From past experience we may safely conjecture that the troops from India, and those previously stationed in South Africa, will not suffer much from typhoid fever. The young soldiers from England, on the contrary, will be exceedingly liable to it. A number of these have been inoculated with an immunising serum, but we have no previous experience to show how far these inoculations may prove effective. It is a well known fact that a person who has had an attack of typhoid fever is generally immune against subsequent attacks, but nevertheless, instances of a second and third attack are by no means rare. Even during convalescence several relapses may occur. This shows that we must rely chiefly on sanitation, on a sanitation judiciously based on the knowledge we now possess of this specific infection.

Filters are not reliable—all drinking water should be boiled, milk should always be boiled and vegetables and meat should be thoroughly cooked. Camps should be erected on elevated and well ventilated sites—cattle should be penned as far as possible from the men—great attention should be paid to any sickness amongst horses and cattle. Infected animals should be slaughtered or isolated, their excrements should be disinfected with chloride of lime and carbolic.

Each individual case of typhoid fever should be considered as a focus of infection and should be at once isolated. The stools and urine of every case should be immediately and effectually disinfected.

Milk of lime, prepared by slaking freshly burned quicklime and stirring up the powder with twice its volume of water, is an efficient and rapid disinfectant for typhoid discharges. This preparation should be freshly made and added to the stool in equal bulk. Thorough mixing and prolonged exposure are necessary to complete disinfection. The thoroughly disinfected stools should then be mixed with earth, and buried in a trench at a distance from all sources of water supply. In the absence of quicklime, corrosive sublimate (1-500) or carbolic acid should be used to disinfect the fæces. All clothing when soiled with fæcal discharges should be moistened with 5 per cent. solution of carbolic acid, and boiled for half-an-hour. Attendants before taking their meals should wash their hands in corrosive sublimate solution (1 to 1,000). These measures should be carried out most scrupulously by those in attendance, not only for their own protection but also to prevent any further dissemination of the infection. In the epidemic of typhoid among the troops in Natal, 1881-2, one half of the men of the Army Hospital Corps were attacked on account of their carelessness.

Latrines should be dug far away from the kitchen, earth should be thrown into them very frequently, and carbolic acid or petroleum should be sprinkled in and around them to keep away flies. Their site should be constantly changed when diarrhoea is at all prevalent, the trenches should be thoroughly disinfected with chloride of lime. It would be advisable to have special latrines for those suffering from diarrhoea.

Horse-dung and the manure from cattle-pens should be carefully collected and burnt to prevent the breeding of flies. Food of every kind should be well protected from them because, in the absence of other suitable substances, flies are likely to deposit their egg in meat, bread or fruit.

Dysentery.—Our knowledge of the etiology of dysentery is still more imperfect than that of typhoid fever. Probably, as in typhoid fever, the enteric specific lesions of dysentery may be complicated by a variety of bacteria from the intestinal flora. Recent researches point more and more to the amœba described by Kartulis as the specific cause of dysentery. The presence of this amœba in the liver abscesses, which frequently complicate dysentery, is another proof of its specific nature.

Against this theory is the fact that the amœba may be absent from the stools of dysenteric patients and from the pus of liver abscesses. I believe this objection to be of no importance whatever. The absence of the amœba in a liver abscess may be satisfactorily explained by phagocytosis. In other diseases we have many similar examples. The bacilli of plague are found simply teeming in the hyperplastic glands of the early stage, but are totally absent when suppuration has taken place. As for its absence from the stools, I infer that the amœba of dysentery is no more than the typhoid bacillus, essentially an intestinal parasite, but that it is eliminated through the liver and intestine, giving sometimes rise to no symptom whatever, and at other times causing very serious lesions in combination with other pathogenic micro-organisms.

It is generally believed, and not without reason,

that the specific agent of dysentery is water-borne. We know also that the disease is very prevalent amongst cattle and sheep. Laveran tells us that in an expedition in Algeria, the French soldiers were seized with dysentery after drinking water which had been fouled by the carcasses of sheep thrown in above the drinking site. The prophylaxis for dysentery is, therefore, the same as for typhoid fever—boiling of water and milk, avoidance of uncooked vegetables and meats, isolation of patients, thorough disinfection of their stools.

(To be continued.)

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

I.

IN your article on the "Absence of some Common Diseases of Temperate Climates in the Tropics," you remark that "no disease stands out more prominently in this respect than does scarlet fever." This is possibly the case, but I think that rheumatism stands out as prominently.

By rheumatism I do not mean the pains which many are in the habit of calling rheumatism, but "a specific disease of the fibrous and serous structures of the motor apparatus," a disease in which, according to Fagge, there are cardiac complications in 50 per cent. to 60 per cent. of the cases, and which is in all essentials the same, whether acute, sub-acute, or chronic.

I wrote to the *Indian Medical Gazette* in December, 1896, expressing this view, and after a three years' residence in the Malay Peninsula I have seen no reason to alter my opinion. I have never, during five years in the tropics, seen a case in which there has been swelling of joints, fever, sour-smelling sweat, the sweating, however copious, not reducing the fever, and a cardiac murmur developing during the fever; nor have I seen a *post mortem* in which there has been found a cardiac lesion, where the previous history has been one anything like that of rheumatic fever. In fact, cardiac vegetations are distinctly rare. If the sub-acute and the chronic forms are in reality the same disease as the acute, I must believe, as

I have not seen the one, that the others also do not exist.

There are, of course, many cases of pain and swelling in joints, and in other fibrous tissues; but for these, as a rule, a cause can be found; and even when no cause is obtainable, there is usually no more reason for calling them rheumatism than for calling them by any other name that may suit the fancy; and by some other name they might do better, as the word rheumatism implies a diagnosis and a line of treatment which may be quite wrong.

I remember one case, as I mentioned in the *Indian Medical Gazette*, in which a cardiac murmur accompanied pain and swelling of joints; salicylates failed; the ova of the anchylostomum were found, and thymol followed by a little iron effected a cure.

My experience may be singular, but if so, it is very peculiar that, out of many cases I have seen, not a single one resembled in the slightest degree the rheumatic fever of England. Others may have seen it in other parts of the tropics, but if so, a statement of the fact with description of cases would, I think, be useful, as many cases get returned as rheumatism which probably have no connection with that disease, and to speak of chronic rheumatism in places where the acute disease does not exist is probably a mistake.

J. TERTIUS CLARKE.

Perak. August 26, 1899.

UNORTHODOX PNEUMONIA.

II.

Apropos of Captain Louis Hughes' note on the absence of Croupous (lobar) pneumonia from Malta (*JOURNAL OF TROPICAL MEDICINE*, vol. ii., p. 42), I am inclined to believe that this is the usual experience nowadays, and that ever since the days of pandemic influenza, ten years ago, the old orthodox straight-forward pneumonia is comparatively rarely seen. The *Indian Medical Gazette* recently (September, 1899) had an editorial on this subject, and Professor Whitla, of Belfast, an article in the *Dublin Journal of Medical*

Science. I can remember seeing plenty of the old lobar pneumonia in India, as typical as the text-books teach, but nowadays this type is very seldom seen in my experience, but rather bronchopneumonia, or catarrhal pneumonia accompanied often by pluerisy, and in bad cases by pericarditis. I think it is time to recognise that influenza now exists in very many places as an endemic disease, breaking out every now and then in a somewhat more epidemic form, but seldom now behaving as it did when re-introduced ten years ago.

W. J. BUCHANAN, B.A., M.B.,

Major I.M.S.

Bhagalpur, Bengal, Oct., 1899.

British Medical Association.

SECTION OF TROPICAL DISEASE.

THE RÔLE OF INSECTS, ARACHNIDS, AND MYRIAPODS IN THE PROPAGATION OF INFECTIVE DISEASES OF MAN AND ANIMALS.¹

By GEORGE NUTTALL, M.D., Ph.D.,

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THOUGH a considerable amount of work has been done on the relation more especially of insects to the spread of infective disease, the subject has not until recently aroused the general interest it deserves at the hands of hygienists. In some recent publications² I have made an attempt to bring together the very scattered literature on this subject, thinking that by so doing suggestions for future work might be given. The facts which have been established by the brilliant researches of Ross, Grassi, Bignami, and Bastianelli with regard to the rôle of various species of mosquitoes in the propagation of malaria have at last aroused more general attention to the part which insects may play in the propagation of disease, and it is probable that the new impetus given by these investigations will lead to fruitful researches in other directions. When we look at the work which has been done on insects as carriers of bacterial agents

of disease we are struck by its relatively small amount, whereas an unlimited amount of experimental work has been devoted to the study of the behaviour of bacteria under various physical and chemical conditions in air, water, soil, food, &c. It is certain that insects may under certain conditions play a most important part, both active and passive, in the propagation of bacterial disease.

The Role of Insects in the Spread of Bacterial Diseases.

(1) *The passive rôle.*—Insects may play a passive rôle as carriers of pathogenic organisms. *Musca domestica* and allied species are chiefly to blame in this respect. Such flies are incapable of "biting," but may from the nature of the food which they seek carry pathogenetic bacteria about on their bodies or within their alimentary tracts, and deposit them on lesions of the mucous membranes or of the skin, or on food. Raimbert in 1869, Davaine in 1870, and many others since, have attributed such a rôle to flies in the propagation of anthrax. Celli in 1888 reported experiments which showed that virulent anthrax bacilli were contained in the fæces of flies which had been fed with material containing these organisms. Proust in 1894 and Heim also in 1894 showed that certain beetles and their larvæ which are found on dried skins might serve to scatter the spores of anthrax. These observers found anthrax spores on the skins which were derived from animals that had died from anthrax, as also in and on the insects named. The fæces of these insects, being light and powdery, are scattered by the slightest current of air. The presence of numerous flies during the plague epidemics has been recorded by some of the older chroniclers. Yersin in 1894, working in Hong-Kong, noticed many dead flies lying about his laboratory where animals which had died from plague were examined. He inoculated an animal with the contents of one fly and noted that it died from plague. The fly was seen to contain bacilli morphologically identical with those of plague. He came to the conclusion that flies might serve as carriers of the germs and play a rôle in the propagation of the disease. Yersin, however, went too far when he concluded from the examination of this one dead fly that all the others had died from plague, as the insects might very well have died from lack of water or coming in contact with disinfecting solutions. In 1897 I made a number of experiments with flies which were fed with the organs of animals which had died from plague. It was found that such flies contained virulent plague bacilli in their fæces for forty-eight hours and even longer when they had received plague organs and then sterile food to eat. In one experiment flies were kept at a temperature of from 12° C. to 14° C., and it was found that they were all alive at the end of eight days. In two other experiments at 14° C. all the flies which had been fed on plague organs were dead by the seventh or eighth day. At a temperature of from 23° C. to 28° C. the infected flies nearly all died within three days. Though it is evident that flies die off more rapidly at high temperatures, these experiments showed that they might live a considerable time whilst carrying plague bacilli in a virulent state. The practical conclusions to which these experiments lead are too self-evident to be mentioned here. From experiments on ants Hankin expressed the belief that these animals might serve to spread the plague by gaining access to the bathrooms in search of water or by defecating there. Nicholas in 1873 relates observations which he made in 1849 at Malta on the warship *Superb* which led him even at that time to conclude that flies might play a very important rôle in the propagation of cholera. Maddox in 1885 observed the cholera spirilla microscopically in the dejections of flies (*Musca vomitoria*) which he had fed with cultures of that organism. Tizzoni and Cattani in 1886 isolated cholera germs from three flies caught in the cholera wards at Bologna. Sawtschenko in 1892 fed flies with cultures and found the spirilla in the fæces after two hours. Simonds in 1892 found spirilla in a fly caught in the post-mortem room at Hamburg. He made a few experiments with flies which had been in contact with the intestines of cholera patients.

¹ A paper read before the Tropical Diseases Section at the meeting of the British Medical Association at Portsmouth on August 2, 1899.

² This subject will be found exhaustively treated in my publications, "*Die Rolle der Insekten, Arachniden (Ixodes), und Myriapoden als Träger bei der Verbreitung von durch Bakterien und thierische Parasiten verursachten Krankheiten des Menschen und der Thiere*" (*Hygienische Rundschau*, Berlin, 1899, vol. ix., 72 pages with bibliography); "*Die Mosquito-Malaria-Theorie*" (*Centralblatt f. Bakteriologie, Abtheil. I.*, 1899, vol. xxv., 40 pages with bibliography); "*Neuere Forschungen ueber die Rolle der Mosquitos bei der Verbreitung der Malaria*" (*ibid.*, vols. xxv. and xxvi., 20 pages with bibliography). See also the forthcoming volume of the Johns Hopkins Hospital Reports, Baltimore, 1899. Reviews of new work in connection with the rôle played by mosquitoes in the propagation of malaria will appear from time to time in the *Centralblatt f. Bakteriologie*.

THE RÔLE OF INSECTS, ARACHNIDS, AND MYRIAPODS IN THE SPREAD OF DISEASES DUE TO PARASITES.

Some Animal Parasites and their Hosts.

Parasite.	Definitive hosts.	Intermediary hosts.	Remarks and authority.
<i>Dypilidium caninum</i> (L.).	Dog, cat, man (rare).	Larva of parasite in dog-louse (<i>Trichodectes canis</i>), dog-flea (<i>Pulex serraticeps</i> , Gervais), and <i>Pulex irritans</i> . Only in fully developed insects.	Melnikoff, 1869; Leuckart, Grassi, and Rovelli, 1888-9; Sonsino, 1888. Successful infection experiments.
<i>Drepanidotea infundibuliformis</i> (Goeze, 1782, Ralliet, 1893).	<i>Gallus domesticus</i> , <i>Coturnix coturnix</i> , <i>Anas boschas</i> , tame duck.	<i>Cysticercus</i> claimed to be in <i>Musca domestica</i> .	Grassi and Rovelli, 1888 and 1892. No infection experiments.
<i>Tænia serpentulus</i> (Schränk).	Various birds.	<i>Geotrupes silvaticus</i> (a dung beetle) claimed to contain <i>cysticercus</i> .	Von Linstow, 1893. No infection experiments.
<i>Hymenolepis pistillum</i> (Duj.).	<i>Sorex araneus</i> .	<i>Glomeris</i> (a myriapod) contains <i>cysticercus</i> .	Villot, 1877. Successful infection experiments.
<i>Hymenolepis diminuta</i> (Rud.).	<i>Mus decumanus</i> , <i>Mus rattus</i> , <i>Mus musculus</i> , <i>Mus Alexandrinus</i> , man (rare, only five cases known).	Various insects: <i>Lepidoptera</i> (usually <i>Asopia farinalis</i> , the parasite found both in caterpillar and butterfly), <i>Orthoptera</i> (<i>Annisolabis annulipes</i>), and two adult <i>coleoptera</i> (<i>Akis spinosa</i> and <i>Scaurus striatus</i>).	Grassi and Rovelli, 1889. Successful infection experiments with feeding <i>annisolabis</i> to rats and man.
<i>Hymenolepis microstoma</i> (Duj.).	<i>Mus rattus</i> ; <i>Mus musculus</i> .	<i>Tenebrio molitor</i> (mealworm).	Grassi and Rovelli, 1889, &c. Successful infection experiments.
<i>Hymenolepis uncinata</i> (St.).	<i>Crocidura aranea</i> .	<i>Glomeris limbatus</i> (a myriapod).	Blanchard, 1891.
<i>Distomum ascidia</i> (van Beneden, 1873).	Bats, <i>Vesperugo pipistrellis</i> , <i>Vesperugo Nathusii</i> , &c.	<i>Cercaria</i> develop in molluscs (<i>Lymnæus stagnalis</i>), bore their way out and penetrate into aquatic larvæ of various insects, remaining in them when they develop into flies.	Von Siebold, 1844; Von Linstow, 1887.
<i>Distomum endolobum</i> (Duj.).	Frogs.	<i>Cercaria</i> bore themselves into a number of aquatic larvæ of insects.	Von Linstow, 1887-1897. Successful infection experiment on <i>Rana temporaria</i> .
<i>Gordius tolosanus</i> (Duj., 1842).	Beetles (16 different species) harbour the second or large larval form of the parasite, the adult form of which is not parasitic. Fish? (Occasional pseudo-parasite of man.)	Aquatic larvæ of numerous insects.	Meissner, 1855; Villot, 1874; Von Linstow, 1893.
<i>Gigantorhynchus gigas</i> (Goeze).	Pig, boar, peccari.	<i>Melolontha vulgaris</i> (May beetle) and its larvæ, <i>Cetonia aurata</i> , <i>Orietes nasicornis</i> , in Europe; <i>Lachnosterna arcuata</i> in North America.	Schneider, 1871; Kaiser, 1887; Stiles, 1891.
<i>Gigantorhynchus moniliformis</i> (Bremser).	<i>Cricetus vulgaris</i> (hamster), <i>Arvicola arvalis</i> , man.	<i>Blaps mucronata</i> (a common beetle) contains encapsuled <i>echinorhynchus</i> .	Grassi and Calandruccio, 1888. Successful infection experiments on white rat and man (Calandruccio infected himself).
<i>Filaria rytipleurites</i> DeLongchamps, 1824).	Rats.	<i>Periplaneta orientalis</i> .	Galeb, 1878. Successful infection experiments.
<i>Spiroptera sanguinolenta</i> (Rud., 1819).	Dog, wolf, fox (?).	<i>Periplaneta orientalis</i> .	Grassi, 1888. Successful infection of dogs with cockroaches.
<i>Spiroptera obtusa</i> .	Mouse.	<i>Tenebrio molitor</i> .	Leuckart.

Parasite.	Definitive hosts.	Intermediary hosts.	Remarks and authority.
<i>Filaria Bancrofti</i> (Cobbold, 1877).	Man.	Mosquito. Spec. ? (Manson), development observed by Sonsino, 1884, in <i>Culex pipiens</i> .	Manson, 1877 ; Lewis, 1878 ; Sonsino, 1883. No infection experiments.
<i>Filaria recondita</i> (Grassi, 1890).	Dog.	<i>Pulex serraticeps</i> , <i>Pulex irritans</i> . Development also observed in a tick (<i>Rhipicephalus</i> <i>ssiculus</i> , Koch).	Grassi and Calandruccio, 1888, 1890. Infection experiments negative.
<i>Pyrosomabigeminum</i> (Southern or Texas cattle fever).	<i>Ixodes</i> or <i>Boophilus</i> <i>bovis</i> . The eggs of infected ticks give rise to young ticks capable of communicating Texas fever.	Cattle.	Theobald Smith and F. L. Kilborne, 1893. Successful infection of cattle by means of young ticks. Development of parasites in ticks not observed. On account of the analogy between Texas fever and malaria parasites, I have chosen provisionally to consider the tick the definitive host.
Proteosoma.	Ross's "grey mosquitoes" resembling <i>Culex pipiens</i> .	Crows, larks, sparrows.	Ross (Calcutta, 1898) describes life-cycle of parasite in mosquito. Successful infection through infected mosquitoes (July 7th, 1898), confirmed by Daniels, 1899.
Crescentic parasites of aestivo-autumnal fever.	Ross's "dappled-winged mosquitoes" at Secunderabad. <i>Anopheles claviger</i> , Nov. 28, 1898, <i>Anopheles pictus</i> or <i>superpictus</i> , <i>Anopheles bifurcatus</i> , <i>Anopheles nigripes</i> , <i>Anopheles pseudo-pictus</i> , June 18, 1899, in Italy.	Man.	Ross, 1898, observed first developmental stages. Grassi, Bignami, and Bastianelli observed complete development and carried out successful infection experiments on man.
Quartan parasites.	<i>Anopheles claviger</i> .	Man.	Grassi, Bignami, and Bastianelli Feb. 5, 1899.
Tertian parasites.	<i>Anopheles claviger</i> , <i>Anopheles bifurcatus</i> .	Man.	Grassi, Bignami, and Bastianelli, April 19, 1899. Successful infection experiment.

After they had been removed the flies were rolled in gelatin tubes after intervals of from four minutes to one hour and a half had elapsed. All the cultures showed colonies of cholera germs. Macrae in 1894, working in conjunction with Simpson and Haffkine in India, observed how flies carried cholera germs to sterilised milk which had been purposely exposed in various places in a prison where cholera prevailed. Buchanan described the occurrence of cholera in a prison at a time when flies were numerous. Cholera appeared in the prison after a strong wind had blown the flies in numbers from the direction of some huts where cholera prevailed. The patients attacked were only those who received their food at the part of the prison nearest to the huts infected with cholera. Regarding typhoid fever, Alessi had isolated virulent typhoid bacilli from the excreta of flies fed on cultures of the typhoid bacillus, and it would seem that flies may infect food after they have fed on typhoid excreta. Flies may also act as passive carriers of infective agents in frambœsia by transferring the specific virus from diseased to healthy persons. Egyptian ophthalmia has long been ascribed to the intermediary agency of flies. The *Hippelates pusio* is believed to disseminate the disease termed "Florida sore-eye," and Dewevre concluded from his experiments that pediculi may also serve as propagators of impetigo.

2. *The active rôle.*—An active rôle may be played by blood-sucking flies in the propagation of bacterial diseases. Experimental evidence is wanting, though clinical writers report a certain number of cases of anthrax, septicæmia, pyæmia, and erysipelas as arising from the bites of flies. In the case of anthrax subjective sensations very frequently lead patients to declare that they have been bitten by an

insect when this is not the case. In many cases infection may result from an infected fly being crushed by the person it has bitten. An active rôle has recently been attributed by clinical writers to blood-sucking insects in plague. Bugs and fleas were supposed to be the active agents here, but experiments made by myself on animals suffering from plague, anthrax, mouse septicæmia, and chicken cholera, all gave negative results. In a large number of experiments where these insects were allowed to bite animals dying from the diseases named and then immediately afterwards transferring them to healthy animals not a single case of infection occurred. Though the dejecta of bugs contained virulent bacilli after twenty-four hours they did not contain them later. In fact, it was shown that both fleas and bugs digest various pathogenic bacteria, which they have taken up with the blood of diseased animals. Simonds in 1898 went so far in his elaborate theorising as to conclude that plague bacilli may acquire a heightened virulence in the bodies of such insects. He assigned an important rôle to these insects in the distribution of plague, but furnished no direct proof of his statements. The entirely negative results of my experiments should weigh more than any gratuitous assumptions. If a bug or flea filled with the blood of a patient containing plague bacilli were crushed and the skin scratched by nails soiled with the blood which it contained, infection might easily occur. It has been asserted that such insects, as well as biting flies, are capable of propagating recurrent fever, the *bouton de Biskra*, frambœsia, leprosy, tuberculosis, and yellow fever, but evidence in this direction is wanted before we can come to any definite conclusion in this respect. In the case of the three last-named diseases the evidence given may well be termed frivolous.

THE RÔLE OF INSECTS, ARACHNIDS, AND MYRIAPODS IN THE SPREAD OF DISEASE DUE TO ANIMAL PARASITES.

Insects, arachnids, and myriapods, while serving as intermediary hosts, may play a passive or an active rôle. 1. They play a passive rôle when they are devoured by a host of the parasite they contain. (*Vide* table.) 2. An active rôle is played when, as in the case of the tick in Texas fever and various mosquitoes in malarious affections of man and animals, they inoculate the parasite into a host by means of their probosces. 3. An intermediary position must be given to mosquitoes in connection with *Filaria Bancrofti* and *Filaria recon dita*, as they infect themselves by sucking the blood of the definitive host.

Insects, &c., without serving as intermediary hosts, may play a passive or an active rôle. 1. A passive rôle is played when insects, &c., transport the eggs of animal parasites and deposit them in food or other substances. Grassi made experiments on flies showing that they might transport the eggs of *Tænia solium*, *Tricocephalus*, &c., and Stiles states that he has seen the eggs of *Ascaris lumbricoides* undergo developmental changes in flies raised from maggots which had been fed with the eggs of the parasites during warm weather. Provided that flies take up the eggs in a sufficiently developed condition they might readily disseminate the parasite by dropping their excretions on food or falling bodily into it. 2. An active rôle is played when insects, &c., carry the agent of disease from one animal to another and inoculate the parasitic, as in tsetse fly disease (Bruce). In the table accompanying this paper will be found a brief summary of our present knowledge of the part played by insects, myriapods, and ticks in the spread of diseases due to various animal parasites.

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

SEVERAL papers of interest to students of tropical medicine were read at the recent Congress of Ophthalmology at Utrecht. The following abstracts of the more important are necessarily of the briefest, owing to considerations of space.

PATHOLOGY OF OCULAR LEPROSY.—M. Franke, of Hamburg, gave the results of his examination of three eyes removed from lepers. In one, the most extensive lesions were found in the ciliary body, which was the site of a quantity of granulation tissue containing numbers of bacilli; bacilli were also found in the cornea (the seat of leprosy interstitial keratitis during life), the root of the iris, and the anterior portion of the sclerotic. In the two other eyes the leprosy manifestations were much more pronounced; the irides and ciliary bodies appeared to be entirely replaced by granulation tissue full of bacilli, which were also present in the retina and choroids. In all three the deeper portions of the eye were unaffected. Franke believes that the theory of the endogenous origin of these cases affords the most reasonable explanation of the lesions seen.

THE TREATMENT OF PANNUS BY PERITOMY.—M. E. Boeckmann strongly advocates a variant of peritomy for the cure of pannus. His procedure consists in resecting a band of conjunctiva 2 to 3 mm. broad and corresponding in length to the extent of the pannus, then scarifying the denuded sclerotic, and leaving the

resulting wound to heal by granulating. The immediate result is an apparent increase in the vascularity of the pannus; within a few days, however, it commences to clear and vision ameliorates *pari passu*. Results are described as brilliant, though occasionally a second or even a third operation is required to render the cure permanent.

PROTARGOL.—M. Darier gives a *résumé* of his experiences in the treatment of various forms of conjunctivitis by protargol. Simple forms, and those due to Weeks' bacillus, the pneumococcus and the staphylococcus, are cured permanently in from three to five days (272 cases). Purulent forms (Neisser's gonococcus) are rapidly ameliorated and ultimately cured by biquotidian applications of 30 per cent. solutions combined with frequent instillation of 5 per cent. collyria, cure being usually complete in fifteen days (37 cases). Conjunctivitis with morax diplobacillus (36 cases) does not yield such favourable results, relapses being frequent. Non-specific blepharo-conjunctivitis (41 cases) is "much ameliorated and often cured." For cases of trachoma M. Darier, like most other observers, finds protargol insufficient, owing to its feeble power of penetration.

ELECTROLYSIS IN TRACHOMA.—M. Coppez describes the routine treatment of trachoma employed at his clinic in Brussels. The patient is chloroformed and the negative electrode (de Wecker's fork) applied to the granular conjunctiva, using a 4 to 5 milliampère current, blood and foam being wiped away with tampons soaked in 4 per 1,000 sublimate solution. The conjunctiva is subsequently touched daily with the same solution. Cases of very thick pannus are treated with jequirity—one application of a 5 per cent. maceration—in addition. The electrolysis treatment is stated to be almost invariably successful. M. Coppez admits that the treatment is severe and very painful. Its routine use certainly does not seem to be justifiable; however satisfactory in speedily destroying granulations it must, I am convinced, lead to subsequent cicatricial contraction and the production of grave and intractable forms of entropion.

MALARIAL EYE AFFECTIONS. *A Correction.*—Dr. D. E. Sulzer, of Paris, writes to me as follows:—"J'ai lu avec beaucoup d'intérêt votre travail sur les 'Malarial Affections of the Eye.' Le chapitre traitant de la 'Malarial Neuritis' contient le passage suivant qui me concerne:—'Sulzer believes that a certain proportion of the cases have malaria as a predisposing cause only, the exciting cause being the indirect action of sunlight.' Mon travail insiste partout sur le fait que la névrite malarienne est une affection directement paludique. Il dit tout le contraire de ce que vous me faites dire. La genèse de l'erreur est la suivante: à la fin de mon travail je décris des cas de *choriorétinite maculaire* (centrale) survenues chez des impaludiques qui ont été exposés à l'action de la lumière solaire, réfléchie par l'eau. Je serais le premier à combattre l'opinion que vous me prêtez si un autre l'émettait. Je prends donc la liberté de vous prier de bien vouloir, par quelques mots adressées au JOURNAL OF TROPICAL MEDICINE remettre les choses

en ordre." I have quoted Dr. Sulzer's own words, as they explain correctly how I fell into the error of attributing to him an expression of opinion entirely differing from that he wished to convey.

M. T. YARR.

Review.

WOUNDS IN WAR. The Mechanism of their Production and their Treatment. By Surgeon-Colonel W. F. Stevenson, A.M.S., Professor of Military Surgery, Netley. Longmans, Green & Co., London, New York and Bombay. Illustrated. 1897.

As was to be expected from the position of the writer, his ample experience and the wealth of material at his command, this work on "Wounds in War" is at once exhaustive and authoritative. Colonel Stevenson discusses modern weapons and projectiles, and the nature and treatment of wounds inflicted by them.

The small modern bullet allows wounds to heal more readily without suppuration; it wounds fewer large blood vessels, and, in consequence, cure is easier and fewer men are mutilated and crippled. "The new arm of small calibre is not only the best, but it is also the most humane, in that it lessens the horrors of war as far as possible (Bruns)."

Injuries to bone have ever proved a subject of serious consideration to the military surgeon, and even at the present day the effects of the modern bullet are variously described. It would seem, however, that at ranges of about 100 yards the splinters from bones are small and loose, whereas with the increase in the range the splintered portions are larger and less displaced. So variable are the effects that in many modern battle fields enemies have been accused of using "explosive bullets" where no such missiles were used. This "explosive" action of the bullet is no modern feature; on the contrary, the older rifle bullets caused a more severe "explosive" effect than the new. Many explanations have been given of the cause of "explosive" injuries by solid bullets, none perhaps are conclusive, but it would seem as if the true explanation is to be found in the fact that the soft parts of the body receive from the traversing bullet a large amount of its energy, and moving outwards "with such a degree of force that they act as secondary missiles and cause further smashing and pulping of the tissue." This, after all, is not an explanation, it is only a statement of anatomical fact.

In regard to the treatment of wounds in war, Listerism has revolutionised the practice. Accepting the fact that it is not usually by the missiles of war that wounds are rendered septic, but rather by (surgically) dirty hands and instruments, it is essential that as a "first aid" dressing on the battle field, the wound should be covered over with an antiseptic dressing at once and without examination. "Dry and absorbent dressing under a bandage which exerts a firm pressure, and, if the wound be so placed to admit of it, a splint to keep the part at rest, should

be applied." For surgeons at the front there is only one line of treatment:—"To occlude the wound, to lay the wounded part in a suitable position on a litter, and to render it provisionally immovable (Reyher)." Each soldier in active service is now-a-days provided with a dressing for immediate use, composed of a piece of gauze, a pad of flax charpie between layers of gauze, a gauze bandage four and a-half yards long, a piece of mackintosh waterproof, and two safety pins. The directions for use are printed upon the inside and outside of the covers, which consist of "an outer cover of cloth sewn, and an inner cover of thin waterproof, cemented so as to make it air-tight."

When bullet wounds become septic owing to long exposure, even "although an interval of seven days had occurred in some cases since the receipt of injury, these septic wounds were converted into aseptic ones by repeated washings with 5 per cent. carbolic lotion and the application of moist carbolic and jute dressings." As an example of the benefits of modern surgery in wounds in war no better illustration can be selected than in cases of wounds of the knee joint. In pre-Listerian days this injury was attended by loss of limb, and frequently loss of life. Since, however, Reyher and Bergmann's results in the Russo-Turkish war of 1878, the treatment of this injury has been bereft of many of its terrors. Reyher treated eighteen "primary aseptic cases" (that is cases not examined at the front) of wounds of the knee joint, simple and severe, at the dressing station and field hospitals. In simple cases he merely purified the mouths of the wounds, covered them over by an antiseptic dressing, and kept the part at rest by splint or plaster of Paris bandage. The severe cases he irrigated and drained, with the result that of the eighteen cases fifteen recovered with movable joints, and three died, a mortality of only 16.6 per cent., as compared with 90 per cent. in the Crimean War and 59 per cent. in the Franco-German War of 1870.

The military surgeon will find this work a store of information, not only as regards the history of his art, but it will prove also a valuable and practical guide, useful in every emergency, as Colonel Stevenson enters into the detail of treatment of every injury of bone, joint, viscus, and blood vessel in the body.

Since Sir Thomas Longmore wrote his classic work, we have had no recent book of the kind, and, with the eventful changes which surgery has traversed in the last few years, it was imperative that a more modern text-book should be produced. This Colonel Stevenson has done, and the book stands without a rival in the literature of any country as a guide to the military surgeon, and as a text-book of the science and art of Military Surgery.

News and Notes.

THE Central British Red Cross Committee have accepted a gift of two cases (about 3,000 rations) of Maggi's Consommé, from Messrs. Cosenza & Co., for the use of the sick and wounded in South Africa.

INOCULATION AGAINST ENTERIC FEVER.—Between 20,000 and 30,000 doses of Professor Wright of Netley's Typhoid Vaccine have been sent to South Africa for the purpose of inoculating the troops. Already some 15,000 men have been treated. Seeing the marked prevalence of enteric fever in previous campaigns in South Africa, the experience to be gained will prove invaluable as a test of the efficacy of the measure. We are strongly of opinion that the prophylactic vaccine introduced by Professor Wright will prove of high value.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

GENTLEMEN,—Will you or, possibly, one of your readers in tropical America very kindly help me to obtain a photograph or coloured picture of Pinta disease? (synonyms—mal del pinta, Tifia Caraate, Quirica, Cute, &c.) None of the ordinary books on tropical diseases give a picture of it, and I am anxious to verify a provisional diagnosis which we have made here in three hospital cases. The disease so far has not been recognised in Egypt or the Soudan.

Yours truly,

Cairo, October 13.

F. M. SANDWICH, M.D.

Communications, Letters, &c., have been received from:—

B.—Capt. W. J. Buchanan I.M.S. (Bhagalpur).

D.—Mr. H. M. Dowler (Dovaes); Major Wm. Dick R.A.M.C. (Netley).

G.—Dr. St. George Gray (St. Lucia).

L.—Dr. W. F. Law (Belfast).

M.—Lieut.-Col. K. Macleod (Netley); Col. T. J. McGann I.M.S. (Bangalore); Dr. Patrick Manson (London).

N.—Dr. F. A. Neal (Mahaica).

S.—Lieut.-Col. N. B. Stevenson (Netley).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.

Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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NOTES FROM LAGOS, WEST AFRICA.—

NOTE VII.—MALARIA AND ANOPHELES IN LAGOS.

By HENRY STRACHAN, L.R.C.P.Lond., M.R.C.S.Eng., F.L.S.
Chief Medical Officer.

IN view of the belief now entertained by an influential and increasing body of investigators that inoculation by infected mosquitoes is a cause, if not the cause, of the prevalence of malarial fevers in certain portions of the globe, it behoves every practitioner interested in malaria to give most careful attention to this view of the subject. For whatever the nature may be of the malarial poison, whatever its origin, and however futile efforts might prove to destroy it, if the means whereby it enters the human system can be decisively shown, there seems no *a priori* reason why it should be more difficult to deal with than such diseases as small-pox or typhoid, as regards prevention.

As to the cause—the well-known malarial protozoon is now practically universally accepted, but reasons why this protozoon attacks human beings, its method of entry into human circulation, and its life history outside the human organism, are still matters requiring closest observation and study. The admirable work of Ross, confirmed in all respects by the great Italian and other authorities on malaria, seems to show that the protozoon is conveyed from one malaria-infected human being to another by the mosquito.

So far, the species of mosquito which acts as the bearer of the disease has been found, in various places, to be confined to the genus *Anopheles*; and farther it has been found that *Anopheles* is not merely

the conveyor of the malarial protozoon, but is an intermediary host in which the life cycle of the protozoon is completed.

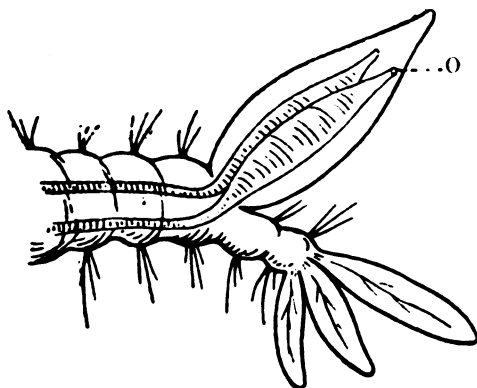
The first question to decide, therefore, seems to be:—*Are species of Anopheles found wherever malarial fevers are notoriously endemic?* The West Coast of Africa is certainly notorious for the prevalence and fatality of its malarial fevers. (I have shown in former "Notes" that natives here suffer and die from these fevers as well as Europeans—a fact well known to observers—though a fatal result is more rare among them.)

Ross' expedition to Sierra Leone has answered question I. in the affirmative, as regards that portion of West Africa, and the purport of this note is to answer it, also in the affirmative, for Lagos.

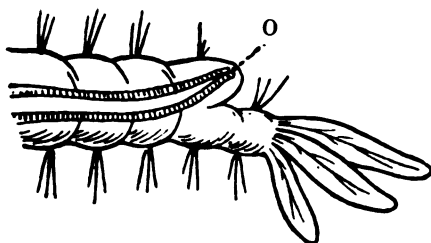
On my arrival here in August this year, I was severely bitten by mosquitoes during the first and second nights. In the mornings I captured many specimens, gorged with blood, and found them all to be of a species of *Anopheles*. On searching the house and capturing a great number of mosquitoes, all proved to be *Anopheles* but one, a *Culex*. I then instituted a search in many different houses, and in all captured *Anopheles*. The next search was for the breeding places; these were found at once. They proved to be (a) small puddles in the streets, resulting from slightly flooded areas, the result of the recent rainy season; and (b) small collections of water of a more permanent character, the result of drainage from surrounding areas, and on which a green vegetable growth is more or less constantly present. These small collections of water and puddles containing myriads of *Anopheles* larvæ, I find scattered in various parts of the town.

The second question of importance in this part of the world is:—*Is Anopheles the only carrier of*

Malaria? Ross' observations appear to a certain degree to answer this in the affirmative for Sierra Leone. But very much work must be done in examination of other suctorial insects, which attack human beings, before this matter can be set at rest.



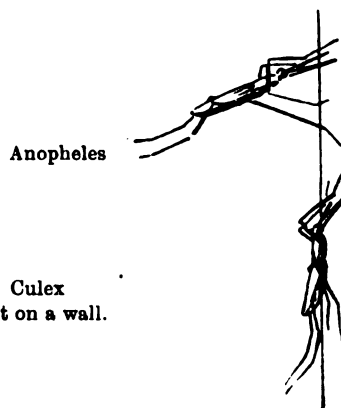
Culex—Breathing apparatus. O = Orifice of trachea.



Anopheles—Breathing apparatus. O = Orifice.



Culex. Anopheles.
Larvæ breathing at surface of water.



Culex
Position at rest on a wall.

I am not yet in a position to give a decisive answer for Lagos, though I have found no zygotes yet in any culex examined. But the following case is of interest in this connection: There had been no cases of fever in the somewhat isolated house where I suffered from anopheles bites, or in its neighbourhood, for some

time, and I escaped without an attack of fever; whereas a gentleman, who arrived in the colony on the same day as myself, and slept in premises where cases of fever existed, and who suffered from anopheles bites on the first few nights, developed a comparatively mild attack of malarial fever early in the third week after his arrival. We both took prophylactic doses of quinine, which may account for his attack having been mild. But infected insects certainly attacked him, while those attacking me appeared to have been non-infected.

At a place called Aro, in the Egba country, where malaria has been prevalent for some months, I found quantities of anopheles.

The third question is:—*What can be done to exterminate or mitigate the mosquito-plague in these regions?* The effect, in malarial countries, of drainage, mechanical, or by water-absorbing trees, is well known. A possible explanation may be, that by it no collections of water are available to the mosquito as breeding places. The action of paraffin oil, in killing mosquito larvæ, has also been well known for many years, and I found it effectual in the West Indies. As anopheles breed in small, and usually shallow, water collections, it is clear that by filling up small depressions where possible, and so preventing collection of water, and by the use of paraffin oil where such prevention is not possible, we may hope to reduce appreciably the number of malarial conveyors in the present; and by gradually destroying the successive broods, and therefore reducing the potential mothers of future generations, get rid, at least to a great extent, of anopheles, so far as the town of Lagos is concerned. It is evident then, that in order to bring about, even to a slight degree, this desirable end, persevering and combined efforts must be made for some considerable period of time. It is necessary, too, that a method of dealing with larvæ in tanks where drinking water is stored, which, while fatal to the larvæ, shall be innocuous to man, should be discovered.

I have been experimenting with many oleaginous substances, and find that the asphyxia resulting from clogging of the breathing orifices of the larvæ, resulting in their destruction, can be obtained if any oleaginous body capable of forming film on the surface of water be employed. Hence, olive oil, especially when emulsified with acacia gum, is effectual though tardy from the fact that the film formed is not continuous, and some of the larvæ may therefore escape for a long time without clogging their breathing apparatus. The addition, however, of a very small proportion of turpentine to the olive oil enables it to spread at once in a thin film, and is more rapidly fatal, while it does not appreciably affect the utility of drinking water drawn by tap from below the surface.

The distinguishing of anopheles from culex both in the larval and imago stages, is, as has been shown by Ross, very simple, and is at once learnt by native assistants. The characteristic attitude of anopheles when at rest, with elevated tail, is due to the fact that the abdomen, thorax, head and "beak" form an almost straight line, making an acute angle with the surface on which anterior legs rest, while the hind pair dangle freely, and is in marked contrast with that of the culex, in which the abdomen and proboscis are

carried at different angles to the thorax when the insect is at rest; and the hind legs are usually turned up over the back, the abdomen lying parallel to the surface on which it stands.

The larvæ of the culex when breathing at the surface of the water float vertically head downward, while those of the anopheles float horizontally. This is due, as microscopic observation shows, to the fact that in the culex, the two main tracheæ are prolonged from the surface of the larva's body into a chamber near the tail, in which they are considerably dilated, in a fusiform manner, before terminating in the small stigmatic orifice; while those of the anopheles terminate almost on the level of the body of the larva, in the same situation, without the buoy-like dilatation.

These points serve to distinguish them apart from the more scientifically accurate peculiarities of the palpi and pigmentation of wings, &c.

So far I have found but one species of anopheles in Lagos, and that in extreme abundance. I saw two species in Sierra Leone when I arrived there with Major Ross and his colleagues, and had the pleasure and advantage of working with them for a day or two. Prolonged observation, therefore, will probably show that other species of the same genus exist here, and in other places on the mainland, and the periods of the year when they are most numerous, which should be toward the end and for a short time after the rains, to coincide with the malaria curve.

In my next note I intend giving a chart to show that this curve coincides here, as it did in the West Indies, with the periods named.

FURTHER RESULTS OF HAFKINE'S ANTI-CHOLERA INOCULATION.

By ARTHUR POWELL, B.A., M.Ch.

THE following tables shew all cases of cholera that occurred since April, 1896, on the estates and villages in my practice, where inoculation has been performed by Mons. Haffkine and myself.

All cases prior to that date have been published in detail from time to time in the *Indian Medical Gazette*, and I gave a *résumé* of my own and all other published cases in the *Lancet* for July 18, 1896.

Judging by the figures one might conclude that the protection conferred by inoculation diminishes as time goes by.

In confirmation of this supposition, I may observe that the most favourable statistics are those of Duna, where the majority of the inoculations were performed in the beginning of 1896.

In this Duna group of houses about half the population was inoculated—at the beginning of the epidemic the non-inoculated were in the majority—but deaths from cholera reduced their numbers, till at the close of the epidemic the inoculated outnumbered them.

There were eleven deaths among the non-inoculated moiety, and but one among the inoculated.

It is perhaps worth mentioning that this case was

inoculated much earlier than any one else in Duna, on February 9, 1895.

Place	UNINOCULATED			INOCULATED		
	Average Population	CHOLERA		Average Population	CHOLERA	
		Cases	Deaths		Cases	Deaths
Karkuri	182	8	8	412	3	3
Kalain	1,033	11	8	1,630	5	5
Kalaincherra	616	8	2	191
Degubber	300	9	6	436	5	1
Duna	61	15	11	59	5	1
River	43	1	1	213	1	1
Total	2,235	52	36	2,941	19	11

The following table shows the incidence of cholera during the same period on those gardens where but a small proportion of the population was inoculated.

Place	UNINOCULATED			INOCULATED		
	Population	CHOLERA		Population	CHOLERA	
		Cases	Deaths		Cases	Deaths
Looba	440	18	10	32
Craigpark	280	6	5	45
Jellalpur	470	32	18	30
Total	1,190	56	33	107
Total of Table 1	2,235	52	36	2,941	19	11
Grand Total	3,425	108	69	3,048	19	11

The figures of the first group of gardens given by me in *Lancet*, July 18, 1896, were 90 cases with 55 deaths among 3,124¹ not inoculated; 8 cases with 3 deaths among 2,730 inoculated.

Thus taking all the cases under my observation up to present date, the proportions are:—198 cases with 124 deaths among 6,549 not inoculated, compared with 27 cases and 14 deaths among 5,778 inoculated.

Had the incidence been the same in both classes, the inoculated would have had 174 instead of 27 cases, and 109 deaths instead of 14.

The mortality among those not inoculated was $7\frac{1}{2}$ greater than among those inoculated.

Method of Inoculating.

It may not be out of place to describe here the method now adopted with Mons. Haffkine's approval,

¹ There was an error of 25 in the population of Duna as given in *Lancet*.

especially as Manson's textbook only describes the method of double inoculation, first with an attenuated comma, then after an interval of five days to five months with one of exalted virulence.

It is known that subcutaneous injection of a comma vibrio recently isolated from a cholera patient causes a local phlegmon and gangrene in the guinea-pig. An immunity to this necrosis is conferred by previously inoculating with an attenuated virus.

Up to 1895, Haffkine applied this method to the human being, using as a "first vaccine" a vibrio which had been isolated in 1884, and attenuated by growth in a current of oxygen.

After an interval of from five days to five months he used as a "second vaccine," vibrios recently obtained from a cholera patient, and exalted in virulence by passages through the peritoneum of the guinea-pig, until a standard dose would kill that animal in eight or twelve hours.

Later, Haffkine thought it possible that the primary inoculation was unnecessary in the human being, and that perhaps even virulent cultures had no local necrosing action on man.

Experiment alone could prove this. With characteristic self-sacrifice, Haffkine asked me to inoculate himself with three times the maximum dose of a very virulent comma.

He had been inoculated two years previously with the primary vaccine, but considered any immunity had passed off after so long an interval.

He would not hear of my getting a *corpus vile*, so I inoculated him with this large dose—very reluctantly—as he was much pulled down by over work and malaria.

Considerable local reaction ensued and high fever, which an examination of the blood showed was partly due to the plasmodium of malaria. No suppuration or phlegmon resulted.

Since then I have inoculated 1,123 persons with virulent, recently isolated vibrios, without using a primary weak "vaccine." In no case has there been any suppuration or other accident.

Briefly described the method is as follows: The whole surface of agar in a sloped tube is inoculated with the comma, and cultivated for from twenty-four to thirty-six hours at a temperature of 40° C.

The whole surface should then be covered with a uniform layer of growth.

Sterilised water is then added to one third the height of the agar, and the growth washed off and suspended in the water by rapidly rotating or shaking, till the surface of the agar is quite clear.

Half a cubic centimetre, about nine minims, is the dose for an average adult.

With reference to Professor Wright's¹ claim to originality in being the first to attempt to produce immunity against disease by means of dead bacilli; I might point out that he himself described Haffkine's original method of inoculation with bacilli killed by carbolic acid, in the *British Medical Journal*, February 4, 1893.

Salmon first conferred immunity in hog-cholera by injecting toxins free from bacilli (*Centraltb. f.*

Bacteriolog., 1887). Wooldridge proved the same for anthrax, and described his results to the Royal Society in 1887.

A CASE OF PARASITIC HÆMOPTYSIS.

By J. PRESTON MAXWELL, M.B., F.R.C.S.

TAM, aged 64, came to me in the summer of 1899, with an internal pterygium in both eyes, on which I operated.

In enquiring into his history I found he had had occasional attacks of hæmoptysis for a year or more, sometimes frequent and sometimes only once a month. He did not look a tubercular subject. Fortunately, while in hospital he had a single attack of hæmoptysis and this gave me the cue to the situation. I then investigated the case with the greatest care and obtained the following facts.

He was born about twelve miles from Changhoo and has never been outside a radius of twelve miles from his home. He has never had any connection with either Japan or Formosa, and none of his relatives suffer from this trouble.

As to his surroundings, he lives about fifty yards from paddy fields, about the same distance from some stagnant ponds, and the same distance from the public w.c.'s, which are simply fæcal pits. His occupation is that of a pig-butcher, and the first attack of hæmoptysis occurred while doing this work.

The history of the illness is as follows:—He was quite well till two years ago, when he began to suffer from piles which he put back with his fingers after each evacuation. About fourteen months ago the attacks of hæmoptysis began. About a teaspoonful of blood would be brought up, and thereafter for a day or two the sputum would be brownish, but this was not at all constant. Sometimes the blood was bright, sometimes dark, and there was never more than this quantity at one time. The attacks usually come on after food, and especially after taking spicy condiments. He has had no special morning cough, and between the attacks is free from cough. At no time has he had pain in the chest and he denies having lost flesh.

Looking at the man he is slightly anæmic, but considering his age, a well preserved man, strong on his legs.

There is nothing special to note about chest or abdomen with the exception that the *superficial* liver dulness extends as high as the fifth rib on the right side. There are no added breath sounds and those present are apparently normal.

There is nothing in throat, mouth or larynx to lead one to suspect that the blood may come from those places.

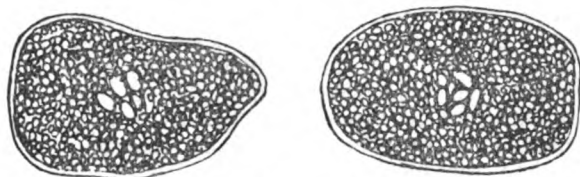
Urine.—Normal, no blood or albumen.

Bowels.—Open daily, mixed piles present, sometimes streaks of blood in stools. In the fæces there are the ova of *ascaris lumbricoides* (many), *trichocephalus dispar* (a few), *ankylostomum duodenale* (very few), and an ovum about which I speak later on.

About three days after entrance he was seized with a fit of coughing while entering my consulting room,

¹ *British Medical Journal*, July, 8, 1899.

and coughed up about a drachm of bright blood. He had just finished his dinner. I at once took possession of this blood and examined it, and without any difficulty found numbers of the ovum described below. They were scattered through the blood, and all presented the same characteristics, varying but little in size and appearance. There were no other ova and no tubercle bacilli were found on examination.



CHARACTERISTICS OF THE OVA.—Light greenish brown; fine double contour; smooth shell; contents granular, with central part more developed; easily ruptured; no operculum made out; 28-32 μ in length, 20 μ in breadth; ovum distinctly transparent.

I tried to hatch some of these but owing to lack of experience did not renew the water sufficiently often, and the whole decomposed and destroyed the remaining ova.

In the fæces I found an ovum, transparent and apparently identical with the foregoing, with the following differences:—

- (a) Lack of brownish tinge.
- (b) No central development of contents, the contents being evenly granular like the external portion of the contents described above.

The patient was treated with thymol and santonin and passed about fifty round worms, but I could find no other parasite in the dejecta. Shortly after the patient insisted on leaving for home.

Comment.—That the hæmoptysis was due to the presence of the parasite whose ova are described here, there can be little doubt. The abnormal characters of the hæmoptysis and the presence of numbers of these ova in the blood expectorated is, to my mind, sufficient evidence of this. I believe the ova found in the stools to be identical, the differences being due to hæmoglobin staining and greater development. The increase of liver dulness is suspicious, and suggests a possibility of a parasite inhabiting the same. Another interesting occurrence is the presence of piles which were returned with the fingers for a year before the hæmoptysis began. Is this a case of auto-infection by means of dried material from the fingers being carried to the mouth and so to the air passages?

POSSIBLE CAUSES OF SICKNESS AMONGST THE BRITISH TROOPS IN SOUTH AFRICA.

By L. W. SAMBON, M.D. (NAPLES).

Lecturer to the London School of Tropical Medicine.

(Continued from p. 106.)

Bilharzia Disease.—The prophylactic measures recommended against typhoid and dysentery will tend not only to protect the troops operating in South Africa against these diseases, but will also greatly reduce the probabilities of their becoming infected by other pathogenic micro-organisms and higher animal parasites such as cestodes and trematodes.

Amongst the trematodes, or flukes, there is one parasite which deserves special attention. This is the *Schistosoma hematobium* (Wienland), generally called *Bilharzia hematobia* (Cobbold) after Professor Bilharz of Cairo, who discovered it in 1851. It produces the disease known as "red water," endemic hæmaturia or better still Bilharzia disease.

Schistosoma is very common along the south-eastern seaboard of South Africa, especially in Uitenhage and Port Elizabeth. It is widely distributed throughout Africa, where it is found in river valleys and low swampy grounds, but it is not limited to that continent as was formerly believed. It has been found in the adjacent islands, on the Arabian coast of the Red Sea and, quite recently, by Sturrock, in the valleys of the Tigris and Euphrates.

We know very little of the zoological distribution of *Schistosoma hematobium*. Sonsino discovered a similar trematode (*S. bovis*) in the veins of oxen and sheep, but it is somewhat larger in size and its eggs are spindle-shaped. Cobbold found another schistosoma in the Sooty Monkey (*Cercopithecus fuliginosus*) and described it as *Bilharzia magna*. Leuckart and Blanchard think the latter may possibly be identical with the species found in man. Lastly, Bomford found the characteristic ova of *Schistosoma hematobium* (?) on microscopic examination of the large intestines of two Calcutta transport cattle destroyed on account of their being suspected of having rinderpest.

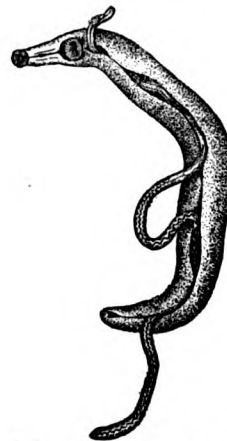


FIG. 1.—Male and female specimens of *Schistosoma hematobium*, enlarged $\times 6$. (After Looss.)

Unlike the majority of trematodes, which are hermaphrodite, in *Schistosoma hematobium* the male and female reproductive organs occur in separate individuals. Both sexes possess an oral sucker and a ventral sucker (the *acetabulum*) placed close to each other. The digestive apparatus consists of a mouth, a short oesophagus, and two intestinal cæca which unite after passing the acetabulum in a single tube. The digestive tract is often found filled with blood globules, thus proving that the worm is hæmatophagus. The two sexes differ greatly in size and appearance. The male measures about 15 mm. in length by 1 mm. in breadth; it is a whitish, flat worm, but presents a cylindrical appearance because its lateral margins curl up ventrally to form a canal, the gynæcophoric canal,

in which the female worm is partially enclosed during fecundation. The surface of the body is covered with minute spines. The *female* is somewhat darker in colour, and may attain 20 mm. in length; she is more slender than the male, and almost cylindrical in shape, being 0.28 mm. broad by 0.21 mm. in diameter. The female is usually found enclosed in the gynæcophoric canal of the male, with her extremities projecting. These parasites are found in the veins of the abdomen, the vena porta, vena renalis, and the venous plexus of the bladder and rectum. The filiform female at the time of parturition is supposed to descend into the smallest branches for oviposition.



FIG. 2.—Egg of *Schistosoma haematobium*, with contained embryo, passed in the urine. $\times 150$. (After Looss.)

The eggs are ovoid to fusiform, 120 to 197 μ long by 40 to 73 μ broad. They have a thin non-operculated shell with a sharp spine usually placed at one extremity, but sometimes laterally. Their segmentation begins and may be completed in the uterus of the parent, and when discharged with the urine they often contain a well-formed embryo.

Several parasites may be found in the same patient. Kartulis in one case found 300 in the portal vein and its branches. Each female deposits many thousands of eggs, an enormous fertility being necessary for the maintenance of the species.

The adult parasites are usually harmless in themselves, but their eggs, in process of elimination, always produce discomfort and sometimes very serious illness. These eggs escape through the genito-urinary system, or through the large intestine, according to the region which the fertile female has selected for oviposition. If the parasite has deposited her eggs in the venous plexus of the genito-urinary system, the characteristic symptom will be hæmaturia, if in the veins of the rectum, the chief symptoms will be those of a proctitis apt to be mistaken for dysentery or piles. In both cases the eggs accumulate in the capillaries, from which they are probably liberated by means of their spines, which, under pressure, it is to be presumed, tear the vessels and pierce the walls of the bladder or rectum, causing more or less hæmorrhage. They are then expelled with the urine or fæces.

While being eliminated through the walls of the rectum, the eggs accumulate in clusters in the mucosa, and give rise to numerous polypoid outgrowths, which may attain 10 mm. to 13 mm. in length. Between these projections the mucosa shows lesions similar to those of chronic dysentery; all the tissues exhibit traces of a chronic inflammatory process; the sub-mucosa is infiltrated with leucocytes, and the muscularis may hypertrophy to three or more times its normal thickness. A similar process takes place in the wall of the bladder, which likewise presents hyper-

trophied patches corresponding to clusters of eggs enveloped by newly-formed connective tissue, and probably undergoing calcification.

The elimination may also take place through the ureters or the seminal vesicles.

When the genito-urinary system is implicated, the urine may contain a quantity of blood, but it gradually becomes clearer after a time, and then only the last few drops contain blood. Micturition is frequently attended with pain, possibly due to the passage of the spined ova. The long continued elimination of eggs through the walls of the bladder usually gives rise to cystitis and sometimes urethritis. A number of complications may supervene, such as urinary fistula, nephritis or hydronephrosis from occlusion of the ureters; the most frequent is calculus. That the calculi result from the presence of the ova of schistosoma is evident from the fact that the latter are invariably found in the nucleus of the stone.

The eggs of schistosoma have been found exceptionally imbedded in the parenchyma of the kidneys, the mesenteric glands, liver and lungs.

The diagnosis of bilharzia disease is easily made by a microscopic examination of the urine or stools to determine the presence of the characteristic ova.

The period of incubation, that is to say the period elapsing between infection and the appearance of the symptoms indicating the beginning of elimination is not definitely known, but Hatch records the case of a patient who remained fourteen days at Suez, and suffered from bilharzial hæmaturia one month after his arrival at Bombay. The disease may last six, eight, ten or even more years, as we know from patients who left the endemic areas of schistosoma soon after being infected. Authors are not agreed as to the longevity of the adult worm. Some believe that it may live ten or more years, others that it probably dies after two or three years, but that its eggs, still containing living embryos, may continue to be eliminated for years thereafter. We may find analogies in support of both theories.

Our knowledge of the life-history of this helminth is very imperfect. In examining the ova of *Schistosoma haematobium* we find that they contain an embryo (*miracidium*), which, like those of other trematodes, is enclosed in a ciliated sheath. Soon after reaching water, the egg-shell suddenly splits longitudinally, and the extruded embryo begins to swim about very actively. Beyond this stage we know nothing further until we find again the adult worm in the veins of a human definitive host. The larval schistosoma very likely undergoes, like other trematodes, successive phases of development in an intermediate host. *Distoma hepaticum*, the common fluke of sheep, has a tiny fresh-water snail (*Limnæa truncatula*) for intermediate host. Within the body of this mollusc it loses its ciliated covering, and becomes a kind of sac called *sporocyst*, the contents of which segments into five or eight bodies called *radia*. These *radia*, having acquired a certain length, pierce the maternal sac, and migrate to some organ, usually the liver. Here they continue to grow, and acquire a higher degree of organisation. The *radia* in dry seasons give birth to daughter *radia*, but in winter they each produce from fifteen to twenty *Cercaria*. The *cercaria* have a flat

ovoid body and a long tail; they soon abandon the body of the mollusc in which they have been reared, and become once more free in the water of ponds and shallow streams. After swimming about for a while they shed their tails and encyst themselves on the stems and leaves of plants, to await being swallowed by their definitive vertebrate host.

Grassi, from observations on *Schistosoma bovis*, is inclined to believe that this trematode may attain the adult form without changing hosts, and Looss has recently expressed a similar opinion with regard to *Schistosoma hæmatobium*. But all experiments have till now proved unsuccessful. However, it is reasonable to assume that *Schistosoma hæmatobium* may gain entrance into our body by means of drinking water, and possibly uncooked vegetables.

We have no specific treatment for bilharzia disease. The symptoms spontaneously subside after a certain number of years. The use of styptics and anthelmintics is based on an unpardonable ignorance of pathological data, and may prove very injurious. During exacerbations of hæmaturia, or of cystitis, the patient should rest as much as possible. Hot baths, hot fomentations, and warm irrigations of the bladder with normal salt solution or boracic water, may prove beneficial.

Plague.—Amongst the diseases which might break out during the campaign in South Africa, we should not overlook the possibility of plague. The disease, in its present extension, has already gained a wide distribution, and, although it be only smouldering at most places, we must not forget that at any moment and in any place it might give rise to a severe conflagration. The conditions of warfare are exceedingly favourable to the spread of the plague, because they upset that sanitary vigilance and care which alone can protect from its inroads. Indeed, most epidemics of plague in Europe have supervened on military operations. An outbreak of the plague at the seat of war would be so disastrous, and might render so futile any attempt to prevent its importation into England, that it is our duty to urge the authorities to take all possible precautions. Cantlie, Simpson and Manson have already given a timely warning, and suggested the main lines of conduct. I can only reiterate what they have said. We must not rely blindly on modern sanitation, which has little to do with plague; we should, on the contrary, consider that, notwithstanding sanitation, we frequently suffer from cruel epidemics of measles, scarlatina, diphtheria and even typhoid. Manson has very justly and clearly explained that the plague is not a water-borne disease, but a rat-borne disease, and that to prevent the plague we should destroy rats. In every single epidemic it has been observed that the rats die by hundreds, that the disease amongst them precedes the outbreak amongst men, and that it constantly follows along the lines of their migration. This all important fact in the etiology of plague was so well known to the ancient Greeks, that in Asia Minor, at the very door of plague, they worshipped Apollo *σμήρως*, that is to say, Apollo the rat, as "the god whose arrows spread the plague."—and, at the same time, as "the destroyer of rats." This awful divinity was represented on monuments treading on a rat. In Roman times, when Esculapius

replaced the more ancient god of medicine, we again find the same fact recorded on a coin of Lucius Verus struck at Pergamum. It represents Esculapius with a rat at his feet, and a small naked figure standing by with arms outstretched.

In studying the history of plague in the light of modern investigation, and especially in connection with the natural history of the muridæ, I have been led to assume that the true endemic region of plague is within the country of Tibet, from which it occasionally spreads into China, India and Persia.

From what we know of other diseases, it is reasonable to infer that the *Bacillus pestis* is not the usual parasite of rats, but of some other animal whose habitat is limited to Tibet, and which has acquired a certain immunity (symbiosis) against this deadly microbe. In this respect it would be interesting to know whether that specially Tibetan animal, the yak, enjoys any immunity from plague.

Although the rat plays so important a role in the dissemination of plague, yet the disease would not spread so widely and so rapidly if it were not for the agency of man. It is the black rat (*mus rattus*) which is more especially the plague rat, because it is the great traveller, the true ship rat.

While recognising fully the paramount importance of the rat, we must not forget that other animals, such as mice, squirrels, monkeys, dogs, cats and man himself, may also become vehicles of the disease.

Fleas, bugs and flies, are likewise very important agents in the dissemination of plague. It is probably through fleas that the disease is most frequently conveyed from rat to rat, from rat to man, and from man to man. The fleas of rats are peculiar to their hosts, but from personal experience I know that under special circumstances they will bite man. Indeed it is not our common house flea (*pulex irritans*), but the flea of dogs and cats (*pulex serraticeps*) that is our greatest tormentor. When a rat dies and gets stiff and cold, the fleas abandon its carcass and crawl about on the floor until they are able to jump on to some other warm-blooded creature. In plague, the greater frequency of the enlargement of the femoral and groin glands, as compared with those of the arm, axilla and neck, is in perfect accordance with the regions most frequently bitten by fleas.

In reference to fleas as carriers of plague it is interesting to notice that besmearing the body with oil is a very old and much valued prophylactic. Tully, in his history of the plague which broke out in 1813 in the Ionian Islands, speaking of those who were employed in attending the sick and burying the dead says: "From the repeated use of oil (which was their sole preservative) their clothes were in a constant state of moisture, and of those who persevered steadily in this practice, few were attacked. But many of these persons subsequently fell victims to their imprudence, by setting at nought the hourly advice they received, seeking comfort by substituting clean clothing for those of their well oiled dresses, which had so long defended them against danger."

The principal measures against plague should be:—

(a) The avoidance of importing grain and other food stuffs from infected places, as rats invariably accom-

pany such cargoes. It is well known that grain ships have been a principal source of plague epidemics.

(b) The destruction of rats in ships, especially when plague infected, or leaving plague stricken localities.

(c) The destruction of rats in towns exposed to infection.

(d) The destruction of stray dogs and cats, which may become carriers of the disease when rats begin to die.

(e) The destruction of fleas in houses and on pet animals.

(f) The isolation of plague patients.

(g) The disinfection of all that comes in contact with the latter.

A CASE OF "FIBROMA MOLLUSCUM AND DERMATOLYSIS."¹

Translated by FREDERIC G. H. EDWARDS (of Mauritius),
M.D., B.A., B.Sc. PARIS, L.R.C.P. LONDON, &c.

M. A., of mixed race (Creole and Madras), born in Mauritius, aged 32; constitution strong, general health good. No trace of similar disease among his parents and relatives; all strong and healthy. Patient was seen for the first time by Dr. Clarenc, who noticed the tumours, which are illustrated by the accompanying photographs (see plate).

These tumours form two main big lumps—one on the face, the other on the chest and shoulder. Besides these, numerous small tumours are found scattered over the trunk and arms. The lump on the face measured 30 cm. from the wing of the nose to its extreme end downwards. It consisted in a thick fold of skin, of triangular shape, having its vertex below, one angle of its base at the left wing of the nose and the other angle at the left ear. From these two last points the tumour was hanging down over the neck and chest.

The anterior margin of the tumour was rounded, smooth, and 4 cm. thick. It was subdivided in smaller lobes by wrinkles. Posteriorly, it involved the ear, which was hypertrophied and considerably drawn down. The skin had here the same aspect as in elephantiasis, especially along the margin of the tumour and behind the ear. At the centre the skin looked as if seen through a magnifying glass. A few hairs were met with here and there; pigmentation was more marked in the sulci, especially in those behind the ear. On the whole, the skin was soft, flaccid, elastic, and movable. Its laxity was remarkable. It allowed the two walls of the fold of skin to separate wide apart from each other, in the same manner as could be done with the scrotum after the puncture of a big hydrocele. The muscles in the region were more or less atrophied; no contraction was elicited in them. The bones could be very clearly defined, as if the thickened skin was the only layer of tissue placed between the observer's fingers and the underlying skeleton.

The patient complained of pain when the tumour

was handled roughly. It was not tender or painful otherwise. The left malar bone seemed hypertrophied, and the angle of the lower jaw could no more be felt, as in normal cases, because the body of the inferior maxillary had been partly rotated laterally on the left side, so that the edge of the teeth were looking much more outwards than upwards. When the patient was seen from the front, the upper tumours showed a sub-mental lobe similar to the others.

On the chest and shoulder another big tumour, divided into three main lobes and having the same characters as that on the face, was noticed. One lobe started at the acromion and extended down to the epigastrium. The second lobe was situated above the former, and covered the supra-spinous fossæ, then passed over the clavicle and joined the first lobe in front of the chest. The third lobe began a little under the supra-sternal notch and hung down as low as 20 cm. below the lowest end of the other lobes, and reached the level of the umbilicus. The different folds of skin were readily lifted up and dragged apart from the body, and their walls used to glide easily on each other just as in the case of the tumour of the face.

All over the body, especially on the chest and arms, were scattered tumours of the skin similar to those already described, from the size of a pin's head to that of a plum; some were flat, others rounded, sessile or pedunculated and soft, their centre being depressed easily with the finger, and giving to the exploring finger a sensation of vacuum as experienced when reducing a small hernia.

The disease began in the patient's youth, and advanced rapidly during the past seven years.

By August, 1893, the pectoral tumour became inflamed, erysipelas set in, and it ulcerated. The patient was then admitted into the hospital. He was treated by moist antiseptic dressings until the inflammation had subsided; afterwards, the tumour of the chest and shoulder only was removed, the patient being unwilling to have all of them entirely removed; this was done with the knife after ligaturing the pedicle.

The results were excellent, as shown in the illustration.

The tumour weighed 1,530 grammes. Section through the skin was difficult, the skin being 2 cm. thick. White fibrous bands were seen macroscopically dividing the skin into compartments of various sizes. Many veins were detected in the tumour. However, the tumour appeared evidently fibrous.

The scar measured 40 cm. after complete cicatrization, but was much longer immediately after the operation.

Microscopical examination was not made.

The author observed that this case, where both "dermatolysis and molluscum fibroma" were associated, is a proof of their being one and the same affection at different stages, and concluded by proposing to call the disease "Multiform Molluscum Fibroma," on account of its various forms.

He adds that this affection is commonly met with among Indians in Mauritius, and he admits of having seen five cases.

¹ Dr. H. Clarenc (of Mauritius) relates the history of the present case in the *Bulletin de la Société Médicale de l'île Maurice*, Nov. 8, 1895.

ON TROPICAL ANÆMIA, AND ITS RELATIONS TO THE LATENT AND TO THE MANIFEST FORMS OF MALARIAL INFECTION.¹

By ALBERT FLEHN, M.D.

Physician of the Imperial Government in Cameroon.

(Continued from page 74.)

CONSEQUENTLY if, as I think I have proved, the poverty of blood which affects regularly both the European and the negro of the coast cannot be accounted for either through the climate, nor as the result of already known diseases, there remains but the supposition that we have to deal here with a *pathological process sui generis which has its specific etiology*; and this seems indeed to be the fact.

In the case of all Europeans who remain in Cameroon, one observes, within a few days or a few months after their arrival (just as in the case of the natives and of the immigrated coloured individuals), almost regularly certain characteristic changes in the red blood corpuscles.

If at the time of the first hæmoglobin reduction, or even later, a little blood be spread in the usual way on the cover-glass, then after hardening in absolute alcohol and staining during eight to twelve hours in Ehrlich's acid hæmatoxylin-alum-eosin solution, the specimen be examined, one may notice that a number of otherwise normal blood-disks show partly either small dark blue points or in part more or less numerous spots of the same colour. Care must be taken to avoid any possible formation of a precipitate, by previously repeatedly filtering the solution, and by immediately closing air-tight the dish when once the specimens have been introduced. It is better, contrary to the general practice, to spread the blood on the cover-glass so as to form several superposed strata. In this way the bodies, which perhaps are only attached to the blood corpuscles, are better protected from injury than if a single layer were spread, and one always finds places in suitable specimens where, besides superposed layers, a single one is present for individual study.

In specimens uninjured during the manipulation the spots usually appear circular. Very often two (more rarely several) touch one another, and assume the shape of a French roll, or short chains (as with diplococcus) are visible.

Sometimes short thick rods are to be seen, often slightly bent, with or without a waist-like constriction. Sometimes smaller spots accompany the larger ones, and by careful focusing it is possible in some cases to recognise that the edge of the small granule projects slightly beyond the margin of the blood-disk.

The size of the granules, usually, varies also in the same erythrocyte. If the granulated blood cells are very scarce, the granules are generally small. They are then often pretty difficult to discover. The more numerous the granulated cells, the more frequent are the large and well-developed granules. The larger

the individual granules the fewer they are in the same blood-cell. Their diameter can attain one-third micron or slightly more; they are, however, mostly smaller. Under certain conditions they occur singly in a blood disk; generally six to twenty or more are present, a smaller number being more rarely met with. Sometimes when the blood is actively decomposing they are to be seen free in the plasma. I was unable up to now to detect any structure owing to the smallness of the granules. When the pigment is so finely divided in the blood-cell that the latter appears as if dusted with coarse flour, it is possible that this is due to a disintegration of the granules. The appearance may then remind one of the dissolution of a nucleus. Such blood-cells always show also the change which Ehrlich described as signs of degeneration. Ehrlich's "Polychromatophilie" otherwise affects but rarely the granulated erythrocytes, although it is very frequent in Cameroon in the case of severe anæmia. The granulated blood-cells appear sometimes slightly larger and spherical, as if swollen.

Methylene blue stains and brings out clearly at least the coarser granules, but the result is not as beautiful as with Ehrlich's hæmatoxylin stain. The carmine stain was uncertain.

I was unable to detect the grains in a fresh and unstained specimen.

There is no visible change in the number of granulated blood corpuscles at the time when, as already mentioned, the amount of hæmoglobin again increases. The number increases on the whole until the first malaria fever breaks out. After its expiration the coarser granules especially are usually strongly reduced, very often they have disappeared altogether. They then grow again in the following period both in number and in size.

If an acute malaria attack remain for a long time without quinine treatment, and if consequently the fever last a number of days, the granules may disappear almost completely.

Chiefly in order to avoid any misunderstanding, I shall denote these bodies, owing to the quality they possess of eagerly taking up nucleus stains, as "*karyochromatophile*" granules.

Often they may be still detected months and years after the individual has left a malaria district, and since the last fever attacks occurred; and already this fact supports the assumption that they are reproduced in the blood.

The karyochromatophilic granules usually disappear more or less completely at every severe attack of blackwater fever. They only reappear again when once the blood begins to be regenerated during convalescence; they then speedily increase in number, and attain their usual size after four or six days. If in simple cases of severe impoverishment of the blood the karyochromatophilic granules be very numerous, one observes regularly, within the next few days, a reduction of 5 to 10 per cent. in the amount of hæmoglobin, which, in some cases, is accompanied with a very noticeable reduction in the number of coarsely granulated erythrocytes. Let it be especially noticed that: *The number and size of the karyochromatophilic granules grow under certain circumstances with the increase of blood, and fall with its decomposi-*

¹ Paper read before the Berlin Medical Society, May 31, 1899.

tion. This, as well as the active regeneration of the blood (which is always present but varying in amount according to the individual), accounts for the case when, on making a *single* examination of the blood, the degree of the anæmia found does not always correspond to the quantity and size of the karyochromatophilic granules, for the result of the examination can alter already within a few days.

The degree of *hyphæmoglobinæmia*, which is by no means always present in anæmic individuals in the tropics, offers the best standard for estimating the decomposition of the blood. According to my investigations the *hyphæmoglobinæmia* is not the result of the washing out of the erythrocytes in the sense of Manson, but rather the consequence of an excessive blood regeneration called forth by the decomposition. Especially in the case of convalescents after black-water fever one may clearly observe that the blood corpuscles contain less hæmoglobin during the first period of blood regeneration.

What importance is to be attached to the karyochromatophilic granules?

I have not detected the change in the blood either of patients suffering from most dissimilar diseases or of apparently healthy individuals, as long as they were not in direct contact with malaria districts. A slightly similar occurrence, however, may be observed owing to the separation of chromatin from decomposing nuclei of erythrocytes, for example in the case of fishes. Ehrlich and Lazarus explain the storing up of chromatin in the red blood disks in the case of anæmia *perniciosa* also through the decomposition of the erythrocyte nuclei. The condition which Bastianelli (according to a communication of Dionisi) observed in the blood of the foetus, and which he considers as an embryonal state of the blood cells, may perhaps be explained in the same manner. In all these cases enough nucleated blood corpuscles are present to warrant the acceptance of Ehrlich's explanation. We have a different state of things in the anæmia now under discussion, for already the frequency of the granulated blood cells excludes any connection with the nucleated ones, the former amounting possibly to 4 per cent. of the normal blood cells, whereas the nucleated ones are to be found only in isolated cases. The similarity may, however, be certainly closer in the case when the karyochromatophilic granules are undergoing, as I have already described, decomposition.

There is not the slightest ground even for supposing that any decomposition process whatsoever due to other quite unknown causes are going on at least during the time *before* the first attacks of malaria or of other diseases. Besides this is contradicted also at a later stage, by the relation existing between the karyochromatophilic granules and the physical changes in the blood.

I am unable to decide whether the spots on the blood disks in the case of the hæmoglobinuric malaria of cattle (as mentioned by Smith and later by Dionisi and also by Liemann), correspond to the changes in the human blood as just described, and this because I had no opportunity of investigating cattle malaria. The explanations offered by the above-mentioned authors (embryonal forms—signs of degeneration) can

hardly hold in the case of the human blood. I shall return to this point later on.

It seems to me that my supposition, that in the case of the karyochromatophilic granules we have to deal with bodies capable of proliferation, *i.e.*, with *organisms*, has a better foundation.

I have already pointed out the relation existing between the changes as described and the malaria attacks, and have come to look upon the karyochromatophilic granules as the *germs of the malaria parasites, which at first increase through division in the red blood corpuscles and cause the latter's destruction, upon which they are partly destroyed in the plasma, and partly remain attached to the blood corpuscles in order to develop in the same way.*

These germs would therefore represent, in a manner, the *primitive forms* of the parasites, seeing that they can continue to exist for months and years in the human blood, and then often produce a hardly noticeable anæmia. Under certain conditions, *viz.*, all those which favour the outbreak of an acute malaria attack, they abandon the "primitive form" for a further modification: *they develop to plasmodia.*

This process, which may most likely be considered as a form of alternate generation, seems to be clearly visible in the stained specimens, if the blood be examined two days before the first attack of fever. At this point one finds the mature karyochromatophilic granules mostly single, or from two to four together, in the red blood corpuscles, and one detects, often only after attentive examination, that in some places a very fine and clearly drawn circle is visible. Sometimes this has hardly twice the diameter of the granule, the latter forming on the circle the "knotty swelling" which has been described already by many authors, and lately also by Koch. According to Romanowski, Grassi, Mannaberg, Liemann and others, the chromatin, or propagation constituent of the parasite, is thus differentiated. Therewith is attained the plasmodium stage.

When the karyochromatophilic granules possess the roll form (probably a stage in the process of division), each half recedes more and more from one another, and we have then the form, already described by another author, in which the parasite possesses two chromatin bodies on the periphery. Exceptionally three have been observed. A marked similarity exists sometimes between the different positions and forms of the karyochromatophilic granules and the chromatin divisions as described and illustrated by Liemann, which are qualified as "constrictions," and which appear shortly before the sporulation of the parasite. Possibly, in this case, there is a certain analogy, inasmuch as the "primitive forms" which correspond to the plasmodia nucleus sub-divide sometimes in the body of the plasmodium itself, sometimes free in the blood cell. A similar process is not an unusual thing in the case of the class of organisms to which the malaria parasites are usually ascribed. However, I do not exclude the possibility that the rod-forms are the result of manipulation.

Not only in the case of Cameroon whites and negroes, but also in the blood of inhabitants of

malaria districts around Rome, did I find bodies corresponding to the karyochromatophilic granules. Moreover I found them in the blood of sailors, who during the last two years had frequented tropical malaria regions, and this holds true whether they had had malaria or not. The individuals examined had been in Rangoon, Calcutta and Mozambique, Guadeloupe, New Orleans, on the Congo and in Lagos, and had therefore acquired the germs in quite different tropical regions. Through the usual kindness of Dr. Lauenstein, I was able to examine them in his hospital during their stay in Hamburg, and was very kindly assisted by Dr. Burmeister, his first assistant.

The few examinations I have been able to carry out until now, owing to lack of time, turned out all positive, although the germs were certainly less numerous and much less developed than in the case of settlers of the West African coast. In their general appearance they resembled more the germs from the *Campana Romana*.

Quite similar granules, besides the parasites, seem to occur in the blood of malaria-struck bats which I obtained from the San Spirito hospital in Rome.

As is well known, Dionisi discovered and described at the beginning of this year a parasite in the bat which resembles most especially the smallest Cameroon plasmodium. He was, moreover, able to ascertain that the bats become exceedingly anæmic under the influence of the disease.

The development of the "primitive forms" seems to me, however, to tally most with that of the so-called *Pyrosoma bigeminum* of cattle malaria (Texas fever), at least from the time when the individual granules occur in the blood cells of the cattle, and with which the karyochromatophilic granules have so often in common the characteristic roll form. Already Smith ascribes this form to a process of division. If this comparison hold good, it follows, contrary to the opinion of Dionisi and Liemann, that the spots in the blood-cells of cattle are perhaps to be considered as parasitic growths corresponding to the "primitive forms" of the parasites in man.

More or less every one who has had experience in dangerous malaria districts, has come to the conclusion that a large number of daily observations cannot be explained through what we know until now of the life history of the malaria parasites; especially, moreover, that there must be a form of this parasite as yet unknown, which is harboured for a long time by the infected individual, and produces the well-known clinical symptoms only after its modification to the so-called plasmodia form. Schellong and Manson have again quite recently expressed most distinctly this opinion. Moreover, it seems rather improbable that the plasmodia form could find favourable conditions for life outside of the human body, seeing that, free, *as such*, in the blood plasma, it is so easily destroyed as it disappears within half-an-hour to eight hours inside the body of the mosquito. The properties of the bodies described by me as the germs of the malaria parasite may perhaps offer an explanation for many points in this respect. I would further wish to state that I explicitly speak of the malaria parasite, for the most recent investigations have

brought forth new and very important points supporting the identity of all forms of parasites, an opinion I have defended already in former writings.

(To be continued.)

PHILIPPINES.

THE WATER SUPPLY OF MANILA.

The greater part of the city of Manila being built but a few feet above high tide, there is never any lack of water, and, as the precipitation is very excessive in the summer months, many houses are provided with tanks to store up the rain water. In addition to this, the city has a fine water system which supplies an abundance of good and pure drinking water free to more than 95 per cent. of the people; the balance pay but a small water rental.

Hydrants are placed conveniently throughout the city supplying water free. Where the water is piped on to private property, hotel, or factory, the water rate is 4 cents a cubic meter daily; and when a large amount of water is used, the rate drops down to 3 cents.

The history of the Water Works is a very interesting one: In the year 1748 San Francisco Cariedo, a Spaniard and resident of Manila, left 10,000 dols. in trust to be used towards constructing a free water supply for the city of Manila. This money in the year 1872, after many vicissitudes, had increased to the sum of over half-a-million dollars. This, together with a meat tax which has been imposed and collected for a number of years previous for that purpose; gave Manila in 1882 as fine and modern a system of water supply as one would expect to find in a modern city in the most progressive country.

The meat tax is still collected, the proceeds of which, with the small amount of water tolls received, pays the running expenses.

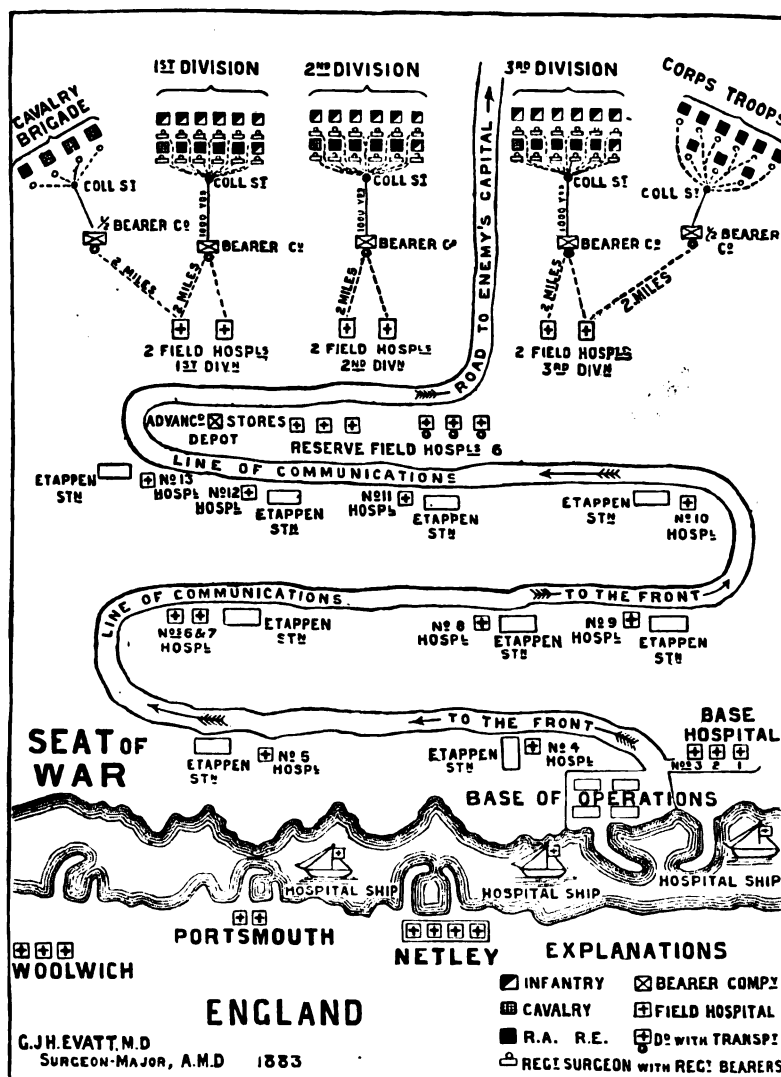
The pumping station is situated about six miles north-east of the city, receiving its water from a mountain stream fresh and pure.

The plant consists of four sets of compound engines, each engine coupling by direct piston connection to the pump. The water is conveyed to the city by a cement conduit for a considerable distance, and the balance by a 26-inch iron pipe. The water in the pipe is under 84 feet pressure, and serves well for street sprinkling, and of some service for fire protection.

The Water Works have been the vital spot at which the insurgents in the recent trouble have again and again concentrated their attacks, they being fully aware that the life and health of the people in Manila are intimately dependent on a pure water supply. The Water Works are now well guarded by the United States troops.

Lieut. William D. Connor is in charge of the Water Works Department.—*Indian Engineering*, July 8.

ENTERIC fever is the chief cause of sickness and invaliding amongst British troops in South Africa. The men affected were those who proceeded to the front before being inoculated with Professor Wright's prophylactic. The protective treatment will therefore be afforded a fair trial, as those who have been inoculated are evidently entering a zone where the disease is prevalent.



PLAN of the AMBULANCE ARRANGEMENTS of a BRITISH ARMY CORPS. Strength: 36,000 men, 12,900 horses, 90 guns, 1,153 waggons.

EXPLANATION OF DIAGRAM.

This Diagram shows each individual Battery, Battalion or Regiment in a British Army Corps, and also the number of units in each Division and Brigade. In the rear of each unit is the Regimental, Battalion, or Battery Surgeon, with his Regimental Ambulance detachment.

The dotted lines show the path of the wounded, *via* the "collecting stations," to the Bearer Companies of each division. Do not confound these with the Regimental Bearers working under the Regimental Surgeons.

Behind the Bearer Companies are the two Field Hospitals of each Division. Massed in their rear on the road leading to the Army are the six reserve Field Hospitals of the Army Corps, not as yet posted to Divisions.

The winding road is the Line of Communications, which may be several hundred miles in length, and which extends from the Base of Operations to the Army in the Front. Along it are placed at the various Etappen, or Halting-stages of the Army, the thirteen Field Hospitals of the Line of Communications.

The winding road is so drawn to save paper.

At the Base of Operations are grouped three or more Field Hospitals, constituting the Base Hospital.

The ships are the Hospital-ships which convey the sick and wounded from the Base Hospital to the English Hospitals at Netley, Portsmouth, Woolwich, &c.

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THE

Journal of Tropical Medicine

DECEMBER, 1899.

MEDICAL PRACTICE IN THE NIGER COAST PROTECTORATE.

THE conditions of practice in the Niger Coast Protectorate are far from being inviting. They are attended with risks which remind us of those encountered by the earlier pioneers in the West and East Indies. It appears that between 1885 and 1893 the doctors of this region were appointed by the merchants trading there, and the number in actual residence was never more than six at one time. The custom was for them to remain on the Coast from a few months to two years, when they went on leave. Notwithstanding this short period of service, out of twenty-seven medical men who practised during the fourteen years, twelve died ; 44 per cent. in this short time is a mortality of an extraordinary ratio, and undoubt-

edly requires attention as to the causes which induce it. An analysis of the deaths show that seven medical men died on the Coast, one was drowned, one committed suicide a week after returning to England, and three died after being a short time settled in England. Of the fifteen that remained alive, nine of them left the Coast early and settled down in England, three of them having been on the Coast only one year, one only six months, and another only three months.

In 1893 a different *régime* was inaugurated, the Niger Coast Protectorate Medical Department was formed, and twelve doctors were appointed between February, 1893, and February, 1894. Of these twelve, five—including the principal medical officer—had been in practice in the Niger Coast before, forming a part of the fifteen mentioned above. It is instructive to pass these twelve men in review, and ascertain what has happened to them. The result is as follows :—One was seized with acute mania and died on the passage home, one died of fever (he had been in practice on the Coast before 1893), one was invalidated home, two retired (one having been in practice before 1893), and seven are still in practice, but of these seven, two of them have been operated on for abscess of the liver. This is a striking record for five years, and bids fair to emulate, or even surpass, that given for the earlier period. Under any circumstance, both periods give a gruesome history of death and sickness similar to those recorded in other tropical lands when visited for the first time by Europeans unaccustomed to the new conditions, and subjected to the hardships which attend on the opening-up of trade routes and carrying civilisation into a land which, so far as the outer world is concerned, has been asleep for ages.

We understand that the medical officers of the Niger Coast Protectorate are entitled to a pension after they reach the age of 55, but it would appear from the statistics that few can ever qualify for that privilege. The pension rules have evidently been based on the regulations which are in force in other places. In India, for example, a Government servant is expected to retire at that age, for long experience has shown that it is quite

exceptional for the State not to have obtained by that time a man's best services. But this can hardly apply to Central Africa, where it is exceptional for the individual to live to that age. Obviously the pension in the two countries must be treated on a different basis. In India the public servant may be worn out at the end of his service, but he is alive, and there is a reasonable expectation of him enjoying his pension for a number of years. In Central Africa, however, the risks to life are greater, and the vast majority, if any, are never likely to reach that age which entitles them to a pension. Under these circumstances, it is only fair and equitable that an adjustment should be made that there may be a fair probability of the medical officer obtaining a pension.

IN connection with the present epidemic of plague in Mauritius, and, considering the vigorous efforts that are being made to stamp it out, its persistency is remarkable. It is interesting and significant to note that Mauritius is peculiarly afflicted by rats: so much so, that the rat-catcher is one of the regular staff on every sugar-cane plantation, and his daily bag is said to range from fifteen to thirty head. The Dutch, who, at one time, attempted to colonise the island, were forced to abandon it, being literally driven out by the armies of rats. The introduction of the mongoose and of rat-killing snakes have been advocated, but effectually objected to by the game preserver and the fowl keeper. P. M.

Article for Discussion.

I.

KUBISGARI.

IS THERE SUCH A DISEASE?

SINCE P. Lucas-Championnierre described this disease in the *Journ. de Méd.*, May 25, 1897, no further light has been thrown upon the ailment he describes. It is unlikely that the disease is confined to the district of Japan, where he first found it, and by this time other observers may

have been able to refute or confirm Lucas-Championnierre's observations, in either Japan itself or in other countries.

The subject was referred to in the *British Medical Journal* of August 7, 1897, but never since has the question been dealt with in any journal.

The disease seems to occur only during the hot season, from May to October, and affects the agricultural population of the villages, more particularly those persons who labour much with horses and cattle. Its geographical distribution is very limited, and it seems to exist only in certain parts of Aomori and Twate. It affects whole families, disappearing in winter, to reappear in spring, affecting the same individuals year after year for as many as thirty years. The character of the disease is intermittent, showing daily attacks more or less short, and which continue unless the patient gives up his work. They may disappear for a few days and then return, seeming to affect the individual when exhausted or hungry, or after having partaken of a dish called Mochi, which is prepared with a glutinous kind of rice. The symptoms are weakness of vision, objects becoming cloudy or dancing before the eyes. This is followed by diplopia, which is a very usual symptom, and photopsia, which is not so common. The examination of the eye shows no change: the pupil reacts to light and accommodation; the ocular movements are unimpaired. The only objective sign which has been discovered in the eyes is some pupillary hyperæmia. These symptoms are accompanied by muscular paresis, more particularly of the upper eyelids and the muscles of the back of the neck. Ptosis or blepharoptosis, which accompanies the visual derangement, is very marked. Paralysis of the neck muscles allows the head to fall downwards and forwards, an effect which has given rise to the name "kubisgari," which means one whose head has fallen. One writer states that he has frequently noticed the peasants' attempt to remedy this condition by a band passing under the chin, and attached to another passing down the back to the waistbelt. There may be also paralysis of the upper limbs with impossibility to close the hand, or of the lower limbs, and accompanied by tituba-

tion, ataxy, and loss of the power of walking, or paralysis of the back muscles, so that the patient is bent double, and unable to lift himself up. There may be paralysis of the tongue, which interferes with speech and other muscles of articulation, causing absolute mutism and inability to open the mouth. In the intervals between the attacks there is no morbid sign if the case is not severe. If it be more marked there may be a certain amount of ptosis and paralysis of the neck in the intervals. It is considered by some, as Miura, that kubisgari is similar to paralytic vertigo observed by Gerlier in the neighbourhood of Geneva. It is also quite possible to mistake "cervical neurasthenia" or "bulbar paralysis without lesion" for this condition. It is suggested that the disease is miasmatic, and certainly its method of recurring in attacks is striking, but there is no enlargement of the spleen, and the blood has been found free from organisms. The fact that it affects those who work hard and among horses is important, and there is evidently some pronounced connection between the disease and stable labour. It seems also that in rural Japan, stables are part of the dwellings, and flies, &c., pass freely from stable refuse to domestic food. What the connection between the two may be is still an open question. Various methods of treatment, such as leeching, blistering, administration of strychnine, iodide, mercury, quinine, &c., have all been tried without effect. Iodide of potash and arsenic appear to have done good in some cases.

It is important to ascertain whether or no this is a specific ailment and if it is right to assign it a place in the nomenclature of Tropical Diseases.

J. C.

SCHOOL OF TROPICAL MEDICINE. LONDON.—The first session of the School is drawing to a close, and most of the medical men who joined in October will soon be leaving to take up their duties abroad. The initial session was, of course, more or less tentative, but the general consensus of opinion, both amongst teachers and taught, is that little, if any, alteration or modification of the curriculum is required. The earnest and close attention of the students showed that the work carried on in the School was interesting and of high educational value. In fact, the first session is pronounced to have been an unqualified success.

II.

DOES RACE INFLUENCE SUSCEPTIBILITY TO VACCINE?

SEEING that the coloured races labour under a special liability to smallpox, would it not be instructive to investigate whether this be accompanied by a correspondingly high receptivity to the related malady, vaccinia? It is surprising to read the authoritative statement, as late as May 30, 1885,¹ of a physician and sanitary officer of thirty years service in India, that his experience in a circle of the North-West Provinces convinced him that a single efficient vaccination was so amply sufficient that he had actually prohibited re-vaccinations. Can this discrepancy from experience in England be due to the high vaccinal receptivity of a people with whom smallpox was an annual visitor? May they have "taken" vaccinia so successfully that renewal of protection was, with their constitutions, unnecessary?

Dr. Seaton, on the other hand, noticed in 1868² the high case rate and relative mortality of European troops in India, and speculated that climate, among other causes, had influenced their protection by vaccination.

Then, again, the Negroes, the most unfortunate of all coloured races as to smallpox, who thus have the strongest reasons for being thankful for vaccination, and the best chances of being impressed by the fate of relatives or comrades who have neglected it, in America at least,³ stand in dread of the protective operation. Is this because it has more perfect success with them than with whites? They might therefore suffer more discomfort than the latter, and have a natural, although not necessarily well founded, ground of alarm. I imagine researches on this point would shed light upon apparently discordant testimonies as to the duration of variolous immunity conferred by vaccine, and possibly yield a fresh corroboration of the truth as to vaccination.

CHARLES G. STUART MONTEATH.

¹ *Lancet*, p. 1014.

² "Handbook of Vaccination," p. 179.

³ *Lancet*, Jan., 1898, p. 136.

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

RHEUMATISM.

(An Article by R. I. Banerjee, B.A., G.B., M.S.L., Pachhadra, Rajputana, in the "*Indian Medical Gazette*," September, 1896.)

MR. BANERJEE classifies rheumatic affections into (1) rheumatism proper; (2) rheumatic pains; (3) pains in persons affected with malaria simulating rheumatism or rheumatic pains; (4) rheumatic fevers; (5) malarial rheumatism; (6) rheumatic malarial fevers; (7) traumatic rheumatism; (8) rheumatism due to blood poisoning. With so distinctive titles one naturally turns to the text to find confirmation of the presence of acute rheumatism (rheumatic fever) in Rajputana. In this, however, one is disappointed. Of 837 cases observed at the Salt Hospital, Pachhadra, Mr. Banerjee remarks, "all such cases were either purely malarial or rheumatism manifested in constitutions badly charged with malarial influences or malaria developed in rheumatic cases."

It is evident that malaria and not a specific rheumatic poison was the ætiological factor present in every case. The so-called rheumatism proper was "the typical form in its acute, sub-acute or chronic varieties," and "all such cases were either in women, or weakly men past middle age, and no children." These facts with the subsequent details of symptoms annul the statement that typical rheumatic fever was met with in any single case. Again, under the heading "Rheumatic Fever," the fact that "the nature of the pain is not usually local but general and diffusive" is sufficient to stamp the name as a misnomer.

To finish with, Mr. Banerjee states that "the multitude of cases treated as rheumatism are not true cases of rheumatism, but symptomatic ailments mixed up with malarial poison." With this conclusion I quite agree. He differentiates cases into "rheumatism proper," and "rheumatic

fever," &c., and describes the symptoms and means of diagnosis of one from the other, whilst he begins and ends with the statement that no rheumatism exists in Pachhadra other than the muscular aching due to malaria infection. Up to the moment we have no reliable statement to justify the belief that rheumatic fever (acute rheumatism) has been met with in the Tropics.

ACUTE RHEUMATIC FEVER IN THE TROPICS.

With reference to your question as to prevalence of acute rheumatic fever in the tropics, my experience leads me to agree with your remarks. In twelve years' experience in India I only remember two cases in which I was disposed to diagnose acute, specific, rheumatic fever. One was in Midnapur, in 1893, and one in January last at Bhagalpur. In the former case I only remember the fact that I hesitated long before making the diagnosis; in the latter case (in January, 1899) the symptoms were in many respects characteristic: the sour sweats, the large, soft pulse, the white furred tongue and the erratic way in which the knee, shoulder and elbow joints were affected. He was also anæmic, so that when the murmur was discovered in the second week of illness it was doubtful if it were organic or not. He recovered slowly in six or seven weeks. Salicylates were of no special benefit, and the heart murmur (mitral systolic) remains. (I examined him again yesterday).

As regards chronic rheumatic arthritis so common at home: this is also, I believe, extremely rare in natives of India. I have at present one patient with the characteristic deformity of the knee joints, but such are very rare in my experience.

Other forms of so-called "rheumatism" are very common in India. Haig (I think) attributes them to prolonged use of cereals as food ("Diet and Food"). Rheumatic pains with and without, or after, malarial fever, are very common. Acute rheumatism with fever of one or two joints is not uncommon, but such are not the specific "rheumatic fever." The army returns give

many cases, both in British and Native Troops, but it may be doubted how many of these are what we are now concerned with, viz., "rheumatic fever," *strictly so-called*.

The question of cardiac diseases in the tropics is worth discussing. Among the thousands of native patients I have examined with the stethoscope I can count up on my two hands the number of cardiac *organic* murmurs (*i.e.*, not functional nor due to anæmia). On the other hand, I have recently made notes of some half a dozen cases among prisoners, of what I have had to return (in accordance with "Nomenclature of Disease") as "disordered action of the heart." In most of these there has been a recent history of influenza or bronchitis. I hope some day to make full notes of them for publication. One certainly sees in no Indian hospital or dispensary heart cases so commonly as in every hospital or out-patients' room at home. Chronic Bright's Disease, again, is very rare in India, though of course we get plenty of albuminuria cases in people suffering from malarial cachexia.

W. J. BUCHANAN, M.B.,
Major, I.M.S.

Magulpur, Bengal,
November 6, 1899.

British Medical Association.

SECTION OF TROPICAL DISEASE.

A FURTHER CONTRIBUTION TO THE STUDY OF MALARIAL EYE AFFECTIONS.

By Major M. T. YARR, F.R.C.S.I., R.A.M.C.

IN the class of cases I propose to deal with in the present communication—mainly, diseases of the conjunctiva, cornea, and iris—the evidence of malarial causation is not so satisfactory as it is in the case of malarial affections of the fundus, as the demonstration of the plasmodia in the affected tissues has so far been lacking. Nevertheless, in many of these cases the suggestion of malarial origin is at least plausible, and in some the clinical evidence in favour of paludism being the *causa causans* appears conclusive. A brief summary of the salient features of these diseases, with references to the few recorded cases, may therefore be of service to those interested in tropical eye diseases, and will complete the survey of the subject commenced in my paper on Malarial Eye Affections, read at the annual meeting of the Association in 1898.

CONJUNCTIVITIS.

At least three different varieties of conjunctivitis have been found connected with malaria, namely: (a) Intermittent ophthalmia; (b) conjunctival injection due to neuralgia of fifth nerve; (c) epidemic conjunctivitis.

(a) *Intermittent ophthalmia* is thus described by Griëssinger:¹ "It is nearly always unilateral, and consists of a more or less marked hyperæmia of the eye, with photophobia, lachrymation, contracted pupil, and frequently swelling of the lids." There is some discomfort, but no neuralgic pain; the symptoms either come on during the attacks of fever in acute intermittents, or replace the paroxysm altogether; in the intermissions the eye is quite healthy; ordinary treatment fails to cure, but it yields readily enough to quinine. Curiously enough, the left eye is nearly always the one affected. That this form of conjunctivitis, accompanying or replacing the paroxysms of acute intermittents, is a real entity is undoubted in view of the many cases collected by acute and cautious observers; that it is directly due to malaria appears almost equally certain. Morton, as far back as 1727,² described a case of tertian fever, apparently cured by quinine, which recurred later in the form of a tertian ophthalmia. Strack,³ in his work on intermittent fever published in 1785, gave notes of a case of quotidian congestion of the left eye, without pain or fever, in a malarious patient. Beylot⁴ saw several cases of the disease in the course of an epidemic of malarial fever at Biskra in Algeria, and Raynaud⁵ has published notes of three cases which he watched carefully, and in which the connection with malaria seems fully established. De Schweinitz, also,⁶ whose authority none can question, states that he has seen instances where the ordinary manifestations of malarial fever were replaced by this intermittent ophthalmia. Many other observations will be quoted in Raynaud's excellent work. Most of the published cases were observed in Algeria and the United States; the disease must be very rare, if not unknown, in the far East, as I never saw an instance in the course of four years' residence in China.

(b) *Conjunctival injection due to neuralgia of the fifth nerve* is very common in malaria and malarial cachectics, as every practitioner in the tropics is aware, but can only be considered indirectly due to malaria. Pain in such cases is severe, and the conjunctival injection comparatively slight.

(c) *Epidemic Conjunctivitis*.—Epidemics of conjunctivitis have been attributed to malaria, notably that in South Carolina in the summer and autumn of 1882.⁷ The concurrence of the disease, however, is probably accidental.

Xerosis.—An affinity has also been traced between epithelial xerosis and malaria, and a connection undoubtedly exists, but probably only inasmuch as xerosis is the local expression of a general malnutrition of which malaria may be one cause.⁸

KERATITIS.

The various forms of keratitis which have been described as malarial may be conveniently grouped under three heads: (a) Dendritic keratitis; (b) Keratitis profunda; (c) Vesicular keratitis (herpes corneæ).

(a) *Dendritic Keratitis*.—Kipp (Newark, U.S.A.) has made a very careful study of malarial keratitis, and there seems little doubt that the "dendritic" form described by him, Van Millingen, and others, is not merely associated with, but directly due to, malaria. Kipp's first description of the disease appeared in 1880; in 1889 he gave his most recent results.⁹ In all he has observed this peculiar form of keratitis in no fewer than 120 malarial patients, in whom the connection between the malarial and corneal affection appeared to be quite clearly established. Beginning, in the course of or after a paroxysm of fever, with photophobia, lachrymation, and supraorbital neuralgia, the characteristic lesion of the cornea soon made its appearance, a peculiar narrow serpiginous superficial ulcer with lateral offshoots, like the skeleton of veins in a lanceolate leaf. Antimalarial treatment cured most cases rapidly; in a few, however, it

had to be supplemented by local applications where the ulcerative process tended to penetrate deeply into the cornea. Kipp's observations were soon confirmed by several other American surgeons, notably Hotz, Miller, Sutphen, and Noyes; the last-named drew special attention to anæsthesia of the cornea, and exaggerated tenderness of the supra-orbital nerves as characteristic symptoms of the disease.¹⁰ The malarial dendritic keratitis described by Van Millingen in 1888¹¹ is evidently the same disease, "fungus-like lesions, with ciliary neuralgia and anæsthesia of the cornea."

(b) *Keratitis Profunda*.—Fuchs, in his *Textbook of Ophthalmology*, states that "intermittent fever in its chronic form of malarial cachexia sometimes results in a keratitis profunda, which is characterised by the absence of marked symptoms of irritation and also by an unusually chronic course." A greyish infiltration of the middle and deep layers of the cornea at or near its centre, comes on very slowly, remains stationary for days or weeks and then subsides without producing any solution of continuity or in fact affecting the superficial layers at all. Under a lens the apparently homogeneous opacity is found to be made up of minute dots and striæ. Lévrier¹² was, I believe, the first to point out the frequent association of this disease with malaria. In one case described by him the patient had suffered severely from malarial fever in Africa; on returning to France the attacks of fever ceased for some time and then returned in the form of shivering and sweating in the early hours of each morning, while at the same time he experienced a slight sensation of uneasiness in the left eye. Seen by Lévrier a month later the upper half of the cornea was found to be opaque, the conjunctiva was injected, and there was slight photophobia; the patient had shivering, followed by a rise of temperature and sweating every morning, exactly the same feverish symptoms as he had suffered from in Africa. Under atropine, fomentations, and quinine in $\frac{1}{2}$ -gram doses twice daily, the eye slowly improved and eventually the cornea was left almost clear. Similar cases have been described by Sedan, Poncet, Landolt, and Javal.¹³ I have seen in Mr. Morton's clinic at Moorfields a case of diffuse central corneal opacity, looking under the magnifying glass like a shower of snowflakes, in a man who had suffered from blackwater fever on the West Coast of Africa. The patient had had rheumatism, but the symptoms most characteristic of rheumatic keratitis, pain, inflammation, and marked photophobia, were wanting. He did well under atropine and small doses of quinine and arsenic, and the infiltration had begun to subside when he ceased attending and we lost sight of him; there was no history of syphilis.

(c) *Vesicular Keratitis*.—Godo¹⁴ in reviewing 40 published cases of herpes febrilis of the cornea says that 18 had occurred in malarials, the corneal eruption being accompanied by herpes of the lips and nose. Tangeman, of Cincinnati,¹⁵ has met with a similar affection amongst malarials which he calls "keratitis bullosa." I hardly think, however, that this herpes corneæ can be considered as directly due to malaria.

IRITIS.

Recorded cases of malarial iritis are not numerous, and in very few is the evidence of malarial origin perfectly satisfactory. While assistant to Mr. Treacher Collins at Moorfields, I saw one case of iritis which seemed to us probably due to malaria. The patient was a discharged soldier, married, with three healthy children. He had had "fever and ague" in Burmah and India. When first seen he complained of violent pain and photophobia in the right eye. The iris was discoloured, and there was considerable ciliary injection, but no synechiæ, and no fever. He stated that he had had three similar attacks in the same eye in Burmah, that they came on during paroxysms of fever, and subsided in a few days' time under quinine. There was no history of gout, rheumatism, or syphilis. He recovered within a week under atropine, and has not returned to the hospital since.

Mr. Collins kindly showed me notes of two other cases he had seen of recurrent iritis in malarials; in both, however, there was a history of gout, and he thought the diagnosis of malarial iritis, though probable, was not absolutely certain. Tangeman's case¹⁶ seems clear enough; his patient suffered from periodic iritis with pain and photophobia, each attack coinciding with a paroxysm of fever; atropine seemed to have no effect, but quinine gave almost immediate relief. M. Péchin, at the recent Congress of the Ophthalmological Society of France (1899) read notes of a case of double malarial iritis.

The patient, a woman aged 48, suffered from tertian ague in 1870; the fever lasted in all several months, but eventually yielded to quinine. During one of the paroxysms her right eye became injected and painful, with discoloration of the iris and immobile pupil; eventually the iritis subsided, leaving a posterior annular synechia. Five years later she suffered again from a severe attack of malarial fever; this time the left eye became affected in one of the paroxysms and several synechiæ formed; she had had no recurrence of iritis or malaria since. Dr. Péchin went carefully into the history and antecedents of the case, and came to the conclusion that the disease was undoubtedly due to paludism.

With reference to this subject Gowers's words (*Medical Ophthalmoscopy*, 3rd edit.) are worth bearing in mind.

Purulent affections of the eye (choroiditis, iritis, etc.) such as are seen in pyæmia, have been described in intermittent fever, but are extremely rare, and some doubt may be felt regarding the diagnosis of the original disease when it is remembered how closely some cases of pyæmia simulate intermittent fever. Even the influence of quinine, on which diagnostic weight is often laid, is not entirely conclusive; for example, in a case of this kind described by Landesberg, although quinine cut short the affection, abscesses formed during convalescence in one toe and forearm.

I hardly see how the formation of abscesses in the case cited invalidates the diagnosis of malaria, as boils and abscesses are very common during convalescence from malarial fever. However, it is always well to remember the possibility of confounding pyæmia with intermittent fever.

CATARACT.

The only instances of cataract attributed to malaria which I have been able to find are two cases recorded by Bagot,¹⁶ of Guadeloupe. The first patient was a mulatto boy of 15 who had a severe bilious remittent fever with gastrointestinal symptoms and coma; this lasted two or three days, and immediately afterwards his sight began to be affected; three months later it was found that he had a soft cataract in each eye. The second patient was a mulatto girl of 16, who also had an attack of grave malarial fever lasting three days; immediately afterwards sight began to fail; nine months later she also was found to have a soft cataract in each eye.

In the absence of more precise details, it seems hardly fair to comment on the above; but judging from the published facts alone, I am inclined to think the coincidence of malaria and cataracts must have been accidental.

ANOMALIES OF ACCOMMODATION.

My friend, Dr. Manson, has shown me notes of a case of monocular ciliary spasm, associated with malarial aphasia, which he saw in Hong Kong; with the cure of the malaria the spasm disappeared. So far as I am aware this is the only case of the kind ever noted; the unilateral character of the spasm is exceedingly curious.

Cases of malarial cycloplegia have also been recorded by Manhaert, Bull, and others.

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ON THERMIC FEVER (SO-CALLED SIRIASIS), WITH SPECIAL REFERENCE TO ITS ALLEGED MICROBIC CAUSATION.

I.—KENNETH MACLEOD, M.D.

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IN the *British Medical Journal*, dated March 19, 1898 (p. 744), Dr. L. Westenra Sambon published a paper entitled, "Remarks on the Etiology of Sun-stroke (Siriasis); not Heat Fever, but an Infectious Disease." In this paper Dr. Sambon contended that so-called thermic fever was not due to heat *per se*, but to a microbe called or recalled into existence and activity by excessive heat under certain circumstances—climatic, local, and personal. He proposed to revive the ancient name "siriasis," as a distinctive term indicating thermic fever, or rather thermic hyperpyrexia. All other cases of heat lesion, of the nature of exhaustion, syncope, or shock, he would place in a separate category, arguing that the symptoms were due rather to ill-health or organic disease of the subject than to the dynamical effect of heat, solar or artificial. Dr. Sambon claims by his rearrangement and fresh interpretation of known facts (for no new facts are adduced) to have removed the disease from the domain of astrology, and straightway fulfils that purpose by calling it after a star. The term "siriasis" has been adopted by Dr. Patrick Manson in his work on tropical diseases, and he evidently inclines also to adopt the hypothesis with which Dr. Sambon has associated the term.

The name appears to me to be a faulty one from various points of view:—

(1) It is more or less mythological; the rising and setting of the dog-star (Sirius) with the sun was anciently credited with the causation of heat and the rise of the Nile as well as with the production of this fever. (2) Heat fever is by no means limited to the dog-days—July 3 to August 11—but, as a matter of fact, is more common at earlier and later periods of the year, at any rate in India. The report of the Sanitary Commissioner with the Government of India for the year 1897 contains good evidence to this effect. (3) A pathological name, when sufficiently justified by facts, is on general principles preferable to a fanciful one. (4) The revived term "siriasis" has been wedded to a hypothesis which rests on a very infirm foundation.

Practically—from the point of view of prevention and treatment—it does not much matter whether the influence of heat is held to act directly or through the intermediacy of an organism. Measures to reduce

heat in the environment and individual must be adopted in either case, and noxious adjuvants have to be avoided or removed on either theory. Still, as names possess some importance—perhaps with some minds much importance—it seems proper to consider whether this new departure is good, and whether the old names of "heat shock" and "heat fever" do not better signify the prejudicial effects of exposure to excessive solar and artificial heat.

Before entering on the merits of the question I wish to draw attention to two spurious antagonisms which have been stated in discussions which have taken place on this subject, namely, (1) an antagonism between dynamical and microbic causes of disease, and (2) an antagonism between toxic and nervous agencies in originating and maintaining increased temperature of the body. As regards the first, it seems unreasonable to attribute to heat such very remarkable power over the life and life energies of microbes, and to deny its influence over the physiological processes of the higher organisms. Besides, even if we allow that microbes and their toxins are the efficient cause of thermic fever, they must still act dynamically; and the question is one of correlation and substitution of energies, which are in a sense antagonistic, while they are likewise mutually sustaining and convertible rather than one of contest and contrast between dynamical and organised agencies, physical and physiological forces. It is vain to attempt to exclude dynamics from pathology, the real issue being their kind and quality.

Similarly, as regards nervous agency in preserving the uniform temperature of the body, the balance between thermogenesis and thermolysis is undoubtedly governed and regulated by the nervous system, whether by the general nervous system or by a special nervous centre is not of much consequence from the present point of view. Toxins disturb the balance, perhaps by producing more heat than can be readily dispersed; perhaps by impairing or disturbing the mechanism which sets in action dispersing agencies. The pyrogenetic influence of toxins is quite consistent with, probably necessarily dependent on, heat-regulating nervous arrangements (which may also be disturbed by other means), and not antagonistic to them.

No positive evidence has been advanced by Dr. Sambon or anyone else regarding the existence of special microbes in cases of thermic fever. It has indeed been shown by Stiles that the injection of blood taken from subjects of this disease into a healthy subject does not induce any pathological disturbance. This would *pro tanto* exclude both infective microbes and toxins from the circulating blood. Dr. Sambon has therefore been compelled, in the absence of proof of the existence of a microbe, to resort to two very treacherous kinds of argumentation—namely, the argument by analogy and the argument by exclusion. The analogies sought to be established concern the epidemiology and the clinical phenomena of heat fever. The relations of the disease to locality and season and its manner of occurrence in communities resemble in a loose sort of way the incidence of some epidemic—microbe-caused—affection; but there are great and important differences, and the constructive evidence thus deduced is very feeble. As regards clinical and pathological analogies, the argument

stated syllogistically would take this form. Diseases characterised by hyperpyrexia are due to toxins elaborated by microbes; heat fever or siriasis is characterised by hyperpyrexia; therefore it is due to a toxin. There is a double fallacy in this syllogism. The major premiss, if general or distributed, simply begs the question. If particular or undistributed it constitutes an example of a middle term undistributed in the major and distributed in the minor, which is faulty. The whole of Dr. Sambon's reasoning may be subjected to this test and found wanting. But there is one analogical argument which was open to him which he has neglected to use—namely, the analogy of the effect of reduced temperature or cold. The chilling of the body is as dangerous as the heating, if not more so. Self-protective arrangements are put in action to conserve heat under chill, just as similar efforts are made to disperse caloric under heat. These arrangements, vasomotor and otherwise, are effected by the agency of the nervous system. General or nervous debility impairs the protective power under cold as under heat. Hygienic and dietetic conditions affect heat conservation as well as heat dispersion. Reduction of temperature—the algid state—is brought about by drugs, frequently by toxins of microbic origin. The phenomena caused by overchilling are peculiar and definite, as are those caused by overheating. The incidence of cold stroke has some resemblance to the incidence of epidemic disease, being in some instances single or sporadic, in others multiple, simultaneous, or quickly successive. Finally, the duration of attacks, many or few, grouped or otherwise, is precisely limited by the duration of excessive cold in one case, and excessive heat in the other. The parallel is almost perfect, and I do not suppose that Dr. Sambon would invoke the aid and intermediacy of a microbe to explain the noxæ, local or general, caused by depressed heat; nor can I recognize the necessity for doing so in mischief due to exalted heat.

The argument by exclusion is equally weak. It takes this form. Excessive heat, solar and artificial can be endured, *per se*, by the human organism with impunity, therefore something in addition is necessary to render it noxious.

The principal exclusions are these:

(1) Experiments on animals, which prove that exposure to excessive solar or artificial heat causes serious or fatal effects, the phenomena of which resemble those of sunstroke in man. The experiments are simply set aside as inapplicable to the case of man. Nevertheless they constitute a large mass of sound research, conducted by such men as Stiles, Kühne, Claude Bernard, Vallin, and Wood, and cannot, in my opinion, be brushed aside in this manner. They undoubtedly establish the fact that sustained superheating proves disastrous or fatal to the animal organisation by producing definite impairments of function and well-marked organic lesions.

(2) Cases of heat stroke, as distinguished from heat fever—of exhaustion, syncope, and shock—are excluded on the ground that they are due to defect in the subject rather than to potency of heat influence. Infirm people are no doubt more prone to succumb to heat than sound people; but it is indisputably true

that perfectly healthy persons are liable to suffer from this sort of sunstroke when the exposure is sufficiently acute and lasting.

(3) The impunity with which excessive solar or artificial heat may be endured for a time under certain circumstances does not assuredly prove that under other circumstances less degrees of it may not do harm. A man may plunge his arm into molten metal under special conditions; but that by no means proves that the experiment is safe under all conditions. Both are cases, in fact, of the exception proving the rule, and, as in the instances of prophetic dreams, presentiments, and so forth, where the rare examples of fulfilment are paraded and the infinitely more numerous cases of non-fulfilment ignored, so marvellous endurance of great heat is apt to be noted, and the much more common instances of damage by heat unconsidered. The distinction between heat shock and heat fever is unquestionably a real one; but there are cases in which sunstroke is the precursor of thermic fever, and in which so-called apoplectic symptoms are succeeded by pyrexial or hyperpyrexial symptoms. This sequence probably occurs more frequently than is apparent: for the circumstances under which heat stroke is sustained are commonly such as to preclude thermometric observation. For this reason it is generally impossible to say whether soldiers stricken fatally on the march or in the field, or civilians in the street, or natives of India taken out of railway carriages moribund or dead, have had hyperpyrexia or not; while men attacked in quarters or in hospital, or persons who come under medical observation and care and linger for days, have their temperatures taken and are found to be hyperpyretic. The main question in this matter is whether derangement or enfeeblement of the heat-regulating centres or heat-dispersing agencies is sufficient under conditions of superheating by exposure to excessive solar or artificial heat to establish hyperpyrexia. This is the view put forward by Hertz and Wood, and which I venture to assert is consistent with both experiments on animals and observation of the effects of heat stroke in man. A consideration (1) of the classes of persons most subject to heat stroke—those, namely, who are most exposed to excessive solar and artificial heat; (2) of the circumstances under which heat stroke is sustained, namely, short exposure to very high temperatures or prolonged exposure to lower but still excessive degrees of heat; and (3) of the conditions pertaining to the subject and environment which exalt susceptibility lends support to the theory. The conditions predisposing to heat fever, whether personal or circumambient are such as: (1) To increase heat production (too much food and drink, excessive exercise, increased metabolism and eremacausis, however arising); (2) to impede heat dispersion (humidity of atmosphere and defective ventilation, hindering evaporation, imperfect action from impaired function or organic disease of skin, lungs, heart, kidneys, or bowels); (3) to impair general and nervous power (debility, alcoholism, fatigue, &c.); and (4) to derange adjustments between thermogenesis and thermolysis (previous or present attacks of fever, prolonged exposure to excessive heat, &c.). The fact that these adjustments are apt under excessive stimulation and exercise to undergo exhaustion or

derangement has not been put forward so prominently as it appears to me its importance deserves. We know that hepatic exhaustion succeeds hepatic stimulation, just as overaction of any organ or function is prone to be followed by failure of activity. Continued exposure to heat impairs the power of resistance to heat, and one of the principal sequelæ of heat stroke or heat fever is an incapacity to endure or resist heat. It seems reasonable, therefore, to believe that fatigue of the heat-regulating nervous centres, as well as of the heat-dispersing agencies, constitutes an essential, or the essential, factor in the production of heat fever.

The questions which present themselves for discussion on the present occasion may be summarily stated as follows:

(1) Is it desirable to adopt the term *siriasis* in preference to and supersession of the term *thermic fever* to denote one of the noxious effects—the *pyrexial*—of exposure to excessive heat?

(2) Is there any evidence regarding the existence of a special microbe or special toxin in cases of heat fever?

(3) Is excessive heat sufficient *per se* to cause heat exhaustion, syncope, or shock in healthy subjects?

(4) Is the doctrine of deranged or exhausted heat regulation, combined with excessive heat production and defective heat dispersion, competent to explain the causation of heat fever?

II.—MAJOR G. M. GILES, I.M.S.

MAJOR GILES said that there was one point which appeared to offer some support to Dr. Sambon's view of a specific rather than a climatic etiology, and that was that heat stroke had a geographical distribution which did not always coincide with a higher air temperature. Sunstroke was almost unknown at the Cape of Good Hope. Troops doing hard work in the field—throwing up fortifications, felling timber, &c., with a blazing sun and an air temperature equal to that of India—wore nothing but their Glengarry caps, and neither officers nor men felt any need for protecting helmets. During these operations, the only case that at all resembled heat stroke was rather of the nature of syncope. It occurred in a sapper working at the bottom of a deep well. Yet, as far as climate went, the conditions in Natal appeared most favourable to sunstroke.

III.—L. W. SAMBON, M.D.

A YEAR and a-half ago I advanced a theory that *siriasis* was an infective disease, and I have since been criticised, perhaps with more pungency than reason, by a few, but on the other hand I have received the support of such authorities as Patrick Manson in England, Professor Firket in Belgium, Dr. Rho in Italy and Professor William Osler in America.

The theory I advocate is not altogether new, because the old Anglo-Indian surgeons who laid the foundations of tropical medicine frequently expressed a doubt whether heat alone could account for the disease.

In reading Hirsch's monumental work on geographical and historical pathology, my attention was aroused

by the fact that the geographical distribution of *siriasis* seemed incompatible with the heat theory. From a careful perusal of the copious literature of sunstroke I found that in the main Hirsch's sketch was correct. *Siriasis* is a widely-spread disease in tropical and subtropical regions, but it exhibits one of the characteristic features of parasitic diseases; its special endemic areas are strictly limited. Like yellow fever, it prevails only in the lowest regions, more especially on coast districts or in the valleys of great rivers. Its altitudinal range is even more restricted than that of yellow fever. Its distribution and prevalence, like that of enteric fever, cholera infantum, and other infective diseases, is seemingly connected with conditions of temperature and moisture. Nevertheless it is absent in regions in which what appear to be the most favourable climatic conditions obtain.

It is as yet impossible to give an exact and complete sketch of the distribution of *siriasis*. Information is exceedingly scanty for some countries; a number of other diseases have been mistaken for *siriasis*, and in statistics it has been frequently classed with cerebral hæmorrhage and other widely-different diseases. But there are salient features in its distribution which are altogether unmistakable. Its constant fluctuating prevalence in certain regions, its constant absence in others which offer even more favourable conditions of heat and moisture. Unfortunately the importance of geographical distribution has not been hitherto sufficiently recognised by medical men, who seem to forget when studying the diseases of man that pathogenic organisms, which after all are plants and animals, are subject to exactly the same laws which govern the disposal of all plants and animals.

Notwithstanding Professor Macleod's assertions to the contrary, a micro-organism has been described as the causative agent of *siriasis* by Cagicol and Lapierre, of the University of Coimbra. It is linear, incurved and slightly constricted in the middle; viewed in the blood it is from 2 to 2.5 μ long, and 0.5 μ in thickness; it stains easily with aniline colours, but not by Gram's method. I have not seen this parasite, and the observations of the two Portuguese doctors have not been confirmed. But the description of a micro-organism assumed to be the specific agent of any disease is not by any means the only reasonable evidence that can be brought forward in support of its parasitic nature. We have no knowledge of the specific organism of small-pox, and yet small-pox is rightly regarded as the type of all infective diseases.

Of course, nothing could be more desirable than the actual demonstration of a specific agent answering thoroughly to Koch's laws, but in the absence of such, I think we can establish the parasitic nature of *siriasis* as satisfactorily as that of other diseases now considered infective, and of which we have as yet no demonstrated parasite.

No discussion is possible on the etiology of *siriasis* unless we are perfectly agreed as to what disease we are speaking of under the name of *siriasis* or *thermic fever*. Most of my critics have confounded *siriasis* with ordinary syncope.

Professor Wood, of Philadelphia, in 1872, gave a very good description of *siriasis*, and clearly recognised its distinction from syncope, which is commonly

called heat exhaustion or sunstroke when occurring in hot weather. Unfortunately, subsequent writers again confounded the two conditions, and some have quite recently described heat exhaustion as a preliminary stage to siriasis. The main characteristics of siriasis are hyperpyrexia, profound coma, contracted pupils, and intense pulmonary congestion. These conditions are widely different from those of syncope, in which temperature is usually normal or below normal, the loss of consciousness is incomplete, the pupils are dilated, and the breathing is easy, though hurried. What is commonly called sunstroke in England during the summer months is nothing more than syncope, which occurs in weakly persons, and especially in those with circulatory unsoundness under the stress of over-exertion or alcohol. It is this condition which occurs so frequently amongst soldiers, who are known to be greatly liable to heart failure. It is mostly in fatiguing campaigns and during the great manœuvres in summer that soldiers fall unconscious on the line of march, especially when oppressed by tight-fitting clothes and heavy accoutrements. A striking example was afforded by the French army at the passage of the Mincio on July 4, 1859, when about 2,000 out of a force of 12,500 men suffered from exhaustion, and 26 died. This example has been erroneously quoted by several authors, who copied from one another, that the French army lost 2,000 men out of a force of 12,000 from sunstroke. This condition was frequently met with in the American Civil War. At the International Medical Congress of Washington, Drs. Sherwood and Goodman stated that cases of siriasis were extremely rare during the war. The majority of cases that went on the army record as sunstroke were simply cases of heat exhaustion in which the heart was mainly involved. Since the war they had opportunity to study the after-history of these cases in the Pension-Bureau, and found that they almost invariably showed some form or other of cardiac disease.

Lieutenant-Colonel McCartie, of the Indian Medical Staff, in a recent article on the relation of the soldier's dress to heat apoplexy, pointed very strongly to the unsuitability of the soldier's dress in India, and showed that it was a great factor in bringing about exhaustion and heat apoplexy. Unfortunately he confounded cerebral hæmorrhage with siriasis.

It is important to bear in mind that whilst siriasis is limited to certain restricted low areas, and in such places is only prevalent during the hot season, heart failure may occur in any place and at any time. Cases of heart failure are quite common in winter, and I remember a number of soldiers falling out faint and unconscious during a long forced march in the winter of 1885 in Naples. The Franco-Prussian War of 1870-71 offered many such examples. I do not mean to imply that heat has nothing to do with cases of syncope in summer, because a comparatively high temperature imposes exceptional work on the heart and circulation, but I believe that impaired resistance is the chief cause of heart failure. It seems surprising that syncope from heart failure should have been confounded with the fever siriasis, but the reason is that both conditions have been given the same name, have been assigned the same cause, and have been consequently considered as different stages of the same

pathological process. As soon as the distinction is clearly made out, siriasis becomes sharply outlined as a distinct disease, with its own unmistakable features. Other conditions and diseases have been confounded with siriasis on account of certain symptomatic resemblances. Thus alcoholic coma, cerebral hæmorrhage, epilepsy, tuberculous meningitis, cerebro-spinal fever, and pernicious malaria have often been mistaken for siriasis.

The name "sunstroke" is derived from the belief that siriasis was caused by the direct action of the sun's rays. This opinion is now obsolete. A number of observers both in India and America have stated that most cases occur at night or in the early morning; in prisons, barracks, and hospitals; in people who have in no way been exposed to the sun's rays. It is for this reason principally that the terms heat stroke and thermic fever were substituted for the older term sunstroke.

The theory that excessive heat is the cause of siriasis is chiefly due to the fact that the disease occurs during the hottest season of the year. This is true, but it is illogical to conclude that because a given fever occurs only in conditions of high atmospheric temperature that fever is necessarily caused by the high atmospheric temperature. There are many other diseases which prevail only during the hot season, but they are not for this reason attributed to heat. If high temperature were the cause of siriasis, we should expect to find the disease wherever and whenever temperature is high; but this is far from being the case. Siriasis is unknown in many of the hottest parts of the world, and, moreover, in its restricted endemic areas it is not specially prevalent in the warmest years, or at the hottest season of the year.

In the United States siriasis becomes imminent during the summer in temperatures varying between 74° and 90° F., but it is unknown in Arizona, in West Africa, in certain parts of India, with temperatures of 120° to 130° F. Stokers on large steam vessels work for as long as four hours at the time in the ill-ventilated and dark stokeholds at temperatures varying from 150° to 180° F., and although occasionally one may be brought up from the furnaces unconscious from syncope, or "stoker's collapse," as it has been called, they never show symptoms of siriasis except in the Red Sea, on the shores of which siriasis is exceedingly prevalent. Indeed, 90 per cent. of all cases in the British Navy occur in the Red Sea, the disease having been contracted while coaling or stopping along its coasts.

There is the widest discrepancy of opinion as to the way in which heat acts in producing the peculiar symptoms and lesions of siriasis. With so obvious a cause as heat one would expect to find the pathology of siriasis as clear and satisfactory as that of a burn. It seems to me that the numerous, varied, and for the most part conflicting, theories advanced to explain the action of heat in producing siriasis tend to prove that we are on a wrong scent.

The numerous theories as to the way in which heat is supposed to produce siriasis are all entirely hypothetical; they do not agree with *post-mortem* findings, and they are not sufficient to explain all facts in the symptoms and natural history of the disease. The

only theory capable of explaining fully the natural history of siriasis is the parasitic theory. It explains the premonitory symptoms which usually precede siriasis, and the relapses which not infrequently follow it; it is in perfect accordance with the symptoms and *post-mortem* appearances of the disease; it explains its epidemic outbursts, the immunity of natives and old residents, the meteorological conditions under which it prevails, and, above all, its peculiar geographical distribution.

Professor Macleod reproaches me for having brushed aside animal experiments. I was obliged to dismiss them in very few words because of the exigencies of space and because of their relative unimportance.

The conditions to which the animals experimented upon were subjected and the resulting phenomena differed widely from those of siriasis. The animals were placed in small closed boxes covered with glass and resting on hot brick flues, or hot-water bonnets were applied to their heads. Stiles immersed them in water at 120° F., and held them by the back of the neck so as to permit respiration until death supervened. In all these experiments death was preceded by convulsions, but never by the characteristic symptomatology of true siriasis. On the other hand, it must be remembered that more recently Naunyn and Welch managed to keep rabbits alive for weeks with an average temperature of 107° F. The animals lay most of the time stretched out, but they took their food greedily and manifested no symptoms of illness. Welch concludes from his own observations and those of Naunyn that a considerable part of the current arguments based upon experiments concerning the injurious effects of high temperature must be revised.

The best proof that these animal experiments have no relation to siriasis was given by Professor Macleod himself when he mentioned Stiles's inoculation experiments. Stiles did not inject the blood taken from subjects of siriasis as Professor Macleod stated, but the blood of animals subjected to artificial heat, and failed to induce any pathological disturbance in the healthy inoculated subject. If Professor Macleod had studied the recent literature of siriasis he would have known that his strongest argument against the parasitic theory of siriasis was null, because several experimenters had proved that the blood serum of patients suffering from true siriasis is exceedingly toxic to animals. Levene and Ira Von Giessen found that 9 c.cm. injected into the ear veins of rabbits killed the animals in less than an hour.

The examination of the blood in siriasis reveals a considerable destruction of red cells and a marked phagocytosis. The destruction of red cells is proved by the presence of pigment in the leucocytes and by the pronounced anæmia following the disease. As the red cells do not disintegrate from heat till the temperature reaches 125·6° to 129° F., the destruction observed in siriasis points distinctly to some toxic element in the blood.

The parenchymatous changes described by Ira Von Giessen after siriasis are similar in every way to those produced by microbic toxins and other poisons. He says that there is no other interpretation open as to the significance of the acute parenchymatous degeneration of the ganglion cells of the brain and spinal

cord than the operation of a toxic substance on these cells. Von Giessen bases on these data a theory that siriasis must be a species of autointoxication.

These facts are of great importance. We have evidently in siriasis a special toxin which, as in other infective diseases, is the cause of the peculiar symptoms and lesions of the disease. This alone would not be sufficient to prove the parasitic nature of siriasis, but taken in conjunction with the geographical distribution of the disease and with the other circumstances already mentioned, it appears to me to be a strong argument in favour of my theory.

IV.—PATRICK MANSON, M.D., LL.D., F.R.C.P.

As a literary performance Professor Macleod's paper is excellent, but as a confutation of Dr. Sambon's contention that siriasis is a bacteria or micro-organismal disease it is of questionable value. Although Dr. Sambon uses an astrological name for a disease which he is endeavouring to show is not an astrological one, and thereby exposes himself to the fun poked at him by Professor Macleod, I yet consider that he has done a distinct service to medicine by rechristening this disease. In my opinion, although I do not consider that he has completely proved his point, Dr. Sambon's arguments in favour of siriasis being a bacterial disease are much more potent than Professor Macleod's arguments to the contrary. The facts of geographical distribution, the definite course, the definite lesions, the relapses, the want of definite correlation to temperature are all in Dr. Sambon's favour. Not one of the facts in connection with siriasis is incompatible with a specific germ cause; many are against its being regarded as the result of high atmospheric temperature pure and simple, and these Dr. Sambon has marshalled for us with a full and critical knowledge of the extensive and confusing literature of the subject.

V.—LIEUTENANT-COLONEL J. P. H. BOILEAU, M.D., A.M.S. (Retired).

Lieutenant-Colonel BOILEAU said that the suddenness with which sunstroke often occurred was a circumstance more in favour of a dynamic change in the system than the development of microbes. Again, with regard to the beneficial effects of cold applied to the body, it seemed more easy to believe in the production of some counteracting dynamic force than a destruction of microbes.

VI.—LIEUTENANT-COLONEL OSWALD WOOD, M.D., R.A.M.C.

Lieutenant-Colonel WOOD said that in Cyprus in the summer of 1878 the British troops were quartered in single bell tents on a treeless plain in the month of July. What the temperature in those tents was he could not say, but in the double-roofed hospital marquees it used to rise to 120° F. during the heat of the day. A large number of cases both of simple heat syncope and of true heat hyperpyrexia, marked by unconsciousness, convulsions, and other symptoms of direct influence on the nervous centres occurred. The nights were pleasantly cool, so that the intense

heat was not continuous. After the troops were huddled and better fed and looked after generally, cases of heatstroke almost entirely disappeared, and since that summer Cyprus had had no unenviable notoriety for such diseases. This would seem to justify the belief that to solar heat and to that alone, without the aid of any specific micro-organism, the numerous cases of heat hyperpyrexia so familiar to the military surgeons who went through that trying experience were due.

VII.—THE PRESIDENT.

Dr. THIN considered it probable that direct sunlight, as distinguished from simple heat, had much to do with the causation of sunstroke, and thought the line between heat exhaustion and sunstroke very clearly drawn. He considered that in many cases sunstroke was at least partly due to the direct effect of light through the eyes.

VIII.—FILIPPO RHO, M.D. Rome.

Dr. RHO (Rome) said that the epidemiological and clinical facts collected by Dr. Sambon from the literature of tropical medicine seemed to suggest that cases of sunstroke were divisible into two classes: the first, true sunstroke, or *coup de chaleur*, met with sporadically, in which the temperature did not go over 40° C.; and the second an epidemic kind of fever with hyperpyrexia, which might be described by the name *siriasis*. In the sporadic cases of real sunstroke, the heat, fatigue, and low health played the first and perhaps the only part; the epidemic cases belonged probably to an infectious disease, but in what way it was impossible to say until bacteriological facts came really to support the new theory.

IX.—JAMES WATSON, M.D.

Dr. JAMES WATSON said that during hot weather persons who for any reason had partaken of an insufficient quantity of food and an excess of alcohol were especially liable to sunstroke. In such conditions the heat centres became readily paralysed, and all power of resistance and accommodation to heat were in abeyance.

X.—LIEUTENANT-COLONEL C. I. MCCARTIE, M.D., I.M.S.

Lieutenant-Colonel MCCARTIE made some remarks on the Cause and Prevention of Heat Apoplexy in the Army, already published in the *Indian Medical Gazette*, June, 1899. He stated that the explanation of the differences in liability to heat apoplexy between civilians and soldiers in India lay in the nature of the dress. As an illustration he said that in August, 1897, he was with a native regiment, marching to the frontier. At one stage sufficient coolies could not be obtained, and part of the baggage had to be carried by sepoy in mufti. Before the end of the march, nearly all the men in the ranks were utterly exhausted, and some of them had heat apoplexy. Their brothers in mufti, though carrying much greater weight, but wearing their loose, light, national dress, were not in the least distressed. They were quite jolly, while the uniformed sepoys were so absolutely prostrated that

they would not have been able to defend themselves if attacked. Dr. Sambon's argument that the soldier died at a temperature which did not affect the civilian, and that therefore his death could not be due to the temperature, but to a disease of an infectious nature, seemed plausible enough until the questions of dress and accoutrements were considered, and the fact that soldiers and civilians did not work under similar circumstances.

Translations.

BLACKWATER FEVER (HÆMOGLOBINURIA).

By R. KOCH.

Translated by P. FALCKE.

(Continued from page 80, October number, 1899.)

No. 5.—Has been in East Africa for three years and often had fever which was easily curable with quinine. During the last week the patient felt unwell, but was still able to attend to his business. The patient finally took fifteen grains of quinine, and, according to his account, only an hour after he had severe rigors and hæmoglobinuria, and was at once admitted to the hospital. His temperature was 40·9° C. when admitted. Next morning there was a decrease of fever, but his temperature rose on the same day to 38·5°, and during the further course of the illness it was subject to considerable fluctuations. The secretion of urine was slight from the beginning; the bloody condition of the urine ceased on the third day. Nevertheless, the quantity of urine did not increase, and occasionally the secretion stopped altogether. The patient during the entire course of the illness was tormented by uncontrollable vomiting. During the first days there was severe icterus, which gradually became less. It was only on the ninth day that death released the patient from his sufferings. The treatment consisted of the administration of morphia and a plentiful supply of fluids. No quinine was given. The collective blood examinations turned out negative.

At the *post-mortem* examination it was found that the epidermis and mucous membranes were icterically discoloured. The spleen was three times the normal size, its substance dark brown, soft, and easily compressible. The kidneys were somewhat enlarged, the cortical substance uniformly light brown, the pyramids striped, dark greyish-brown.

No trace of malaria parasites could be discovered in the sectional preparations of the spleen, liver, and kidneys. There was no deposit of pigment in the spleen and liver. The straight urinary tubules were mostly obstructed by coagulated hæmoglobin; the convoluted tubules were much stretched by the engorged liquid contents.

No. 6.—Goanese. Had come from Bombay to East Africa at the same time as No. 4. He had, according to his account, not suffered with fever in India, but

since his arrival in Africa had repeatedly had attacks, for which he took quinine with good results.

Before his present illness he took fifteen grains of quinine in consequence of feeling unwell, and a short time afterwards he was attacked by violent rigors, succeeded by heat and the secretion of blackish-red urine. He was at once taken to the hospital. The quantity of bloody urine was inconsiderable, but from the second day to the evening of the fifth day, when death supervened, there was almost complete anuria. The continuous vomiting, the restlessness, and the anguish of mind tormented the patient in a horrible manner. Malaria parasites were never found at the blood examinations.

The *post mortem* yielded the same changes as No. 5. Here also no malaria parasites could be found in the spleen and liver. The deposit of pigment in the endothelium of the spleen and liver, so characteristic of malarial disease, was likewise lacking. In this case the kidney convoluted tubules were mostly obstructed by flakes of hæmoglobin.

Besides the facts herewith recorded, which alone suffice to prove that blackwater fever is not malaria, there are a few other circumstances which point to the same conclusion.

There is first of all the attack itself, which only possesses an apparent conformity with an attack of malaria, but which allows of considerable deviations being recognised on careful examination.

As a rule blackwater fever, when it does occur at all during the course of an attack of malaria, is connected with tropical fever. The attack then is never known to begin with a rigor, but at most with slight shiverings. Blackwater fever, on the other hand, sets in, without exception, with a very severe and long-lasting ague (for an hour and over). The rise of temperature corresponds, and is more rapid than in tropical fever. The temperature of the former generally sinks earlier and more suddenly than that of the latter. In consequence of this it possesses much more similarity to the tertian temperature than to the tropical fever temperature.

There follows here two histories of cases which came under observation in the hospital from the commencement; this does not occur very frequently, for generally the patients only come under treatment after the ague is over and the temperature has already reached its maximum.

No. 7 (observation of Dr. Doering). The patient has been in W. Africa for one and three-quarter years. During the last few days he had intermittent fever. On the morning of the attack of blackwater fever he felt well. At 11 o'clock he took 15 grains of quinine. At 12.30 a rigor, lasting one and a-half hours, set in, with vomiting; the urine was dark brownish-red, with a great deal of hæmoglobin; slight icterus. At 4 p.m. his temperature had already risen to 40.5° C. Treatment with morphia, bits of ice. No quinine. Mild attack and quick convalescence.

Dr. Doering could find no malaria parasites in the blood of the patient. In Berlin also nothing was discovered in the preparations placed at our disposal.

No. 8 (observation of Dr. Doering). Patient has been in W. Africa for one and a-half years. Nine months ago he had a slight attack of blackwater fever.

He fell ill with malaria. Temperature towards mid-day 40° C., and at 6 p.m. it fell to 36° C. The patient took 15 grains of quinine, and three hours after (at 9 p.m.) he had a severe rigor. Over night he vomited dark green, sticky material; the small quantity of urine passed was densely black. Towards morning the skin of the patient looked lemon-coloured. There were malaria parasites in the blood (a small number of ring-shaped, unpigmented parasites were also found in Berlin in the corresponding preparations). Severe course. Continuous vomiting. Complete anuria. Death on the tenth day of the illness.

No malaria parasites could be found in the blood taken from the patient on the fifth and ninth days.

(To be continued.)

Recent Literature on Tropical Medicine.

TROPICAL OPHTHALMOLOGY.

RETINAL SEQUELÆ OF TROPICAL FEVERS.—The October number of the *Chicago Ophthalmic Record* contains an interesting paper by Dr. W. K. Rogers, entitled, "Report of Seven Cases of Retinal Sequelæ of Diseases affecting U.S. troops in Cuba and Porto Rico." The cases came under the writer's observation soon after the return of the American troops from the Cuban campaign. All the patients had suffered from "some of the various febrile disturbances encountered by the troops during their stay in Cuba and Porto Rico." A very brief abstract of each case will be of interest.

Case I.—Edematous condition of retina of left eye, with impaired vision and contracted field. The oedema gradually subsided, leaving vision and field unchanged. Patient had had yellow fever a month before. Blood normal.

Case II.—Invalided for greatly impaired vision of both eyes; marked malarial cachexia. In the right macular region were numerous glistening white points, some with surrounding pigmentation. In left a few similar points at the macula, and just below it a moderate-sized crescent-shaped hæmorrhage. Urine normal. Blood—hæmoglobin, 42 per cent.; red corpuscles, 2,600,000; white, 6,000. The hæmorrhage became absorbed, but without improvement of vision.

Case III.—Patient had typhoid fever three months before in Cuba; since then noticed vision impaired. In the macular regions of both eyes were a number of sago-like patches in the deeper portion of the retina; some were scantily pigmented, none showed any appearance of atrophy. Blood examination showed merely simple anæmia. Urine normal.

Case IV.—Impaired vision after typhoid fever and dysentery; pronounced anæmia. Marked swelling around right disc; macular region bounded by a sharply-defined ring of distinct hyperæmia, enclosing an area slightly less than one disc diameter, of a comparatively pale colour, in which the macula appeared as a bright red spot—"such a picture as one might expect in a partial thrombosis of a central vein."

In the left œdema round disc, with venous engorgement and a few radiating opaque striæ around macula. Under general treatment the œdema disappeared, and vision slightly improved; opaque striæ remained in each eye. Urine: trace of albumen; no casts. Blood: hæmoglobin, 65 per cent.; red cells, 3,100,000, white normal ratio.

Case V.—Impaired vision after yellow fever. Proliferative striæ in temporal region of each retina adjacent to small vessels, showing whitish reflex and extending, 1 to 6 D, into each vitreous. Blood and urine normal. Vision became much improved on correcting an error of refraction.

Case VI.—Pronounced malarial cachexia following pernicious attack, with hæmatemesis and melæna. In each macular region was a dark granular area, throughout which was a fine net-like, yellowish exudation; in the right, beyond this area, to the temporal side and somewhat upwards, were three patches of exudation in the deeper layers of the retina. Vision much impaired. Blood: hæmoglobin, 60 per cent.; red corpuscles, 2,600,000; white, 12,000. Urine normal. General health improved. Vision and fundus changes remained unaltered.

Case VII.—Well-marked neuro-retinitis in left, following malarial fever. Blood and urine normal. History of syphilis eighteen months before.

The symmetrical macular changes seen in Case VI. are extremely curious, and in all probability specifically malarial. It seems unfortunate that no search was made for the plasmodium in any of the cases. The neuro-retinitis in Case VII. probably owed its origin to syphilis, and not to malaria.

RETINITIS PROLIFERANS DUE TO MALARIA.—The *Annales d'Oculistique* for November give an abstract of a case of "double detachment of retina with consecutive retinitis proliferans," described by Tornabene, an Italian observer, and attributed by him to malaria. The patient had had several attacks of malarial fever, and had never suffered from syphilis. The case is an interesting one, but the evidence of malarial origin hardly strikes one as satisfactory, although no other cause could be discovered.

As my duties with the Siamese Government necessitate my spending part of the present, and nearly all of the following month in Russia, I fear I shall be unable to contribute this column to the January number of the JOURNAL.

M. T. YARR.

[The Editors regret the temporary absence of Major Yarr, to whom they are indebted for many excellent articles on Tropical Ophthalmology.]

Foods, Drugs, &c.

THE MAGGI CONSOMMÉ.—By the enterprise of M. Maggi, and under the immediate direction of several Swiss medical men of repute, a condensed vegetable soup has now for some time been before the public. The ingredients of the Consommé are contained in small gelatine capsules, and it only requires the addi-

tion of three-quarters of a pint of boiling water to each tube to produce an excellent beverage. For travellers bulk is everything, and this preparation meets all requirements in that direction. It is important to know that the gelatine tubes are not affected by tropical heat. Maggi's Consommé is pleasant, easily digested and very reasonable in price. We can with confidence recommend them to all travellers proceeding to tropical countries.

CARBOLIC ACID IN THE TREATMENT OF PLAGUE.

By Dr. J. M. ATKINSON.

Principal Medical Officer, Hong-Kong.

THE patient, aged 30 years, a sanitary inspector, was admitted to the Government Civil Hospital, Hong-kong, on June 9, 1899, with the following history. He had been very hard-worked and had been exposed to the trying climate of the colony at the worst season of the year. On June 6, in the course of his duties, whilst conveying a plague patient down the staircase of 34, Bridges Street, to the ambulance, he slipped and abraded the skin over his left elbow. The following day he felt pain in the left groin. He complained of fever on the evening of June 8, and on the morning of the 9th was ordered to the hospital.

On admission at 7.30 p.m. there was distinct tenderness in the glands of the left femoral region which were obviously enlarged. He was ordered 2 grs. of calomel, and 15 grs. of migranin were given to relieve the headache. As he complained of want of sleep at 11 p.m. $\frac{1}{2}$ of a gr. of morphia was given hypodermically. On June 10 plague bacilli were detected in blood. Three grs. of carbolic acid in the form of a pill were given every four hours. At twelve midday the temperature was 105.6°; 4 grs. of phenacetin with 1 gr. of citrate of caffeine were now given. It was then felt that if carbolic acid was going to do any good it must be pressed, so at 4 p.m. one oz. of the following mixture was ordered to be taken every two hours: carbolic acid (Calvert's pure), 12 grs.; syrup of ginger, 1 drachm; and chloroform water to 6 ozs. Four doses were given that evening. On June 11 the mixture was resumed at 7 a.m. and between that time and 8.30 p.m. six doses were given. On June 12 the temperature had fallen to 101°. The medicine was continued every two hours. On June 13, the patient complained for the first time of slight pain on passing urine; the dose of the mixture was reduced one-half and given every four hours. The temperature had fallen to 99.8° and the pulse was 72. The patient's temperature was practically normal on June 15, the dose of the mixture was reduced to two grains of carbolic acid thrice daily; this dose was continued to June 26, when strangury was again complained of, the urine was dark coloured, and the carbolic acid was now discontinued. All this time the bubo had been maturing and on June 27 it was incised. The diet during the acute stage was liquid and stimulating, six ounces of brandy being given in the twenty-four hours. This was reduced on June 16 to two ounces daily. (*Lancet*, Dec. 9, 1899).

Correspondence.

ON TROPICAL ANÆMIA.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—You made a mistake concerning my paper in your Journal. You wrote: "Avoiding sunlight causes anæmia." That is not at all my opinion. Avoiding sunlight causes a pale face, and the pale face may produce the *wrong* impression of anæmia, although investigation of the blood proves that there is *no anæmia at all*. I consider this mistake as a very important one. Please will you kindly correct it by a notice in the next number of your Journal.

I send you another paper about "blackwater fever," to translate and to publish in your Journal. You see I have not the same opinion about "blackwater fever" as R. Koch.

Yours truly,
(DR.) PLEHN.

October 18, 1899.

THE NEEDED BOOK ON THE MOSQUITO.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—With reference to your remarks as to the necessity for the preparation of a work on the above subject, it may not be out of place to mention that I have been engaged on the preparation of a book of the kind for the last year, and that it is now so near completion that I hope to see it through the press in the course of a month or two. The literature of the subject is so scattered, and in so many languages, that the work of collecting and translating the published descriptions has proved far more difficult than I had expected: but for this the work would have ere this been issued, but as nearly all the published descriptions have now been collected, the promptness of its issue will now depend entirely upon my publishers.

G. M. GILES, M.B., F.R.C.S.
Major I.M.S.

Plymouth,
November 18, 1899.

PRICKLY HEAT.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—Mr. Pearse and I appear to have misunderstood each other on the subject of prickly heat.

There are one or two points which I think require a little explanation. (1) The affection called prickly heat is *not* a *seborrhœa*. If the skin in a case of prickly heat be examined with a powerful lens before the patient has irritated it by scratching, it will be seen to be studded with minute vesicles containing a clear fluid, and if these vesicles be punctured, the fluid that exudes will be found to be watery and not oily. (2) Allow me to repeat my remark, "where the perspiration evaporates rapidly, or can be drained away by absorbent underwear, there is little or no prickly heat." When it is so excessive that it cannot be drained away, or that the exposed surfaces still remain bathed in perspiration, the cause of the affection is not removed. (3) Lanoline, well rubbed in, is undoubtedly the best treatment if the patient cannot be removed to a cooler climate. My remarks on the laundry question were elicited by Mr. Pearse's concluding statement that "it must be *very* freely used." I did not understand by that that he meant a certain limited quantity carefully rubbed into the skin. My experience of the use of Lanoline extends over about thirteen years, and hardly a day passes that I do not use it in my practice. I appreciate its valuable properties, and should be very sorry if I were obliged to discard it altogether.

ST. GEO. GRAY, M.B., B.Ch. (Univ. Dub.),
Colonial Assistant Surgeon.

Castries, St. Lucia.

THE SURGERY OF THE JAWS ON THE BATTLE-FIELD.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—Some time since, in a letter to your valuable paper, I advocated the institution of a Post-Graduate Dental Course, to be taken up by those medical men who enter the service of the Army and Navy. Such a progressive step by the authorities would, I consider, satisfy a need felt, and unequivocally expressed, by officers in both branches of the Service in times of peace; but the necessity for the knowledge and practice of dental surgery by Army surgeons, in campaigning, is apparent to the least intelligent when facts are traversed. The authorities have now recognised the wisdom of sending specialists to the seat of war in our South African campaign, by sending out medical men skilled in abdominal surgery; why do they not carry out this good intention to the full, and have all branches of surgery represented by specialists at the front? For instance, it is not necessary to point out the incalculable amount of good which would result from ophthalmic surgeons being sent, but as my work is dental surgery and space is valuable, I will confine myself to my own sphere, and plunge in *medias res*, by saying that although Army surgeons may be theoretically conversant with the methods of the application of interdental splints, they are not supplied with the necessary appliances and tools to enable them to do so.

To have some idea of the magnitude of a dental surgeon's work in a war, we may glance at the report on the American War. The Surgeon-General of the American Army reported in November, 1865, that from the commencement of the war to October, 1864, out of 4,167 wounds of the face, there were 1,579 fractures of the facial bones. The Crimean returns from April 1, 1855, to the end of the war, show 533 face wounds, of which the bones were injured in 107 instances. Now, with the multiplication of destructive weapons and the introduction of terribly explosive shells, wounds must be of greater number and variety, and if there be progress in the art of destruction and wounding, surely there should be a corresponding progress in the means of saving life and relieving pain on the battle-field. In maxillary and mandibular fractures, the surgeon on the field will apply a four-tailed bandage, and that is all, and lucky it will be if the wounded soldier does not experience non-union or union with malposition by the time he arrives at the base, where a dental surgeon may be found. If the War Office will not send dental surgeons to the front, I ask why do they not insist on their surgeons equipping themselves to enable them to apply the various interdental splints? Can they do it now? Emphatically I say they cannot. In fracture of the mandible neglect of treatment or a badly-adapted splint will be likely to produce non-union. In this fracture, when the teeth are present, a Hammond is a most serviceable splint, and a description of it, together with the Gunning splint, might not be out of place here. For a Hammond fill two dental trays with softened bees-wax, and press them into the upper and lower maxillæ; remove them, and fill these impressions with plaster of Paris, allow it to set, then warm the bees-wax, and

remove it, when you have a model in plaster of both upper and lower jaws. The lower model should now be cut through with a fret-saw at the point or points of fracture, and the pieces articulated with the teeth of the model of the upper jaw, and kept by means of plaster of Paris in this their natural position. Soft iron wire is now bent, and adapted at the necks of the teeth, all along the lingual and labial sides, the ends being joined by soft solder; when this is done, the fragments in the mouth are brought into position, the splint placed over them, and the teeth fixed to it by means of iron binding wire passed round the wire of the splint, then between the teeth, round the opposite wire, and then back again between the teeth, care having been previously taken to free the teeth of all tartar. This interdental splint can be very quickly made if the simple appliances are at hand; but, for argument, are they at hand at Ladysmith at this moment? The probabilities are there is not such a thing as a dental tray in the place, and how many fractured jaws Dundee produced will be interesting to know, all of which have been, *volens volens*, detained in Ladysmith without proper treatment. The Gunning splint consists of vulcanite caps joined together by supports, and constructed to fit both the upper and lower jaws. These caps are filled with gutta-percha, which should be thoroughly softened, the displacement reduced as much as possible, and the splint forced into position. A four-tailed bandage is then applied, and the jaws are then practically fixed into the splint by the outside bandage. There are also the Hayward and the Hern splints. The choice of a splint will depend to a great extent upon the requirements of each individual case. For a fracture occurring in a mouth containing firm teeth, so that each fragment contains a few, a Hammond splint is best. These two splints, I consider, would be found more generally indicated in maxillary fracture in the battle-field.

GORDON HOOPER, L.D.S.

Communications, Letters, &c., have been received from:—

- A.—Dr. R. E. Adamson (Labuan).
 E.—Dr. F. G. H. Edwards (London).
 F.—Mr. R. Fitch, R.N. (Cape of Good Hope).
 G.—Major G. N. Giles (Plymouth); Dr. St. George Gray (St. Lucia); Dr. J. C. Graham (Sumatra).
 H.—Dr. A. H. Hanley (London).
 J.—Major J. M. Jones, R.A.M.C. (Bombay); Mr. Hampden Jackson (Liverpool).
 L.—Dr. W. F. Law (Dublin).
 M.—Mr. H. S. Muir, Surg.-Genl. (18, Victoria Street).
 N.—Dr. H. A. Alford Nicholls (Dominica).
 S.—Dr. Hy. Strachan (Lagos); Dr. Sandwith (Cairo).
 W.—Dr. M. J. Wright (Perak); Dr. John Weddick (Hawaiian Islands); Prof. G. Sims Woodhead (Cambridge).

EXCHANGES.

Annali di Medicina Navale.
 Archiv. für Schiffs u. Tropen Hygiene.
 Archives de Medicine Navale.
 Australasian Medical Gazette.
 Boletín de Medicina Naval.
 Boston Medical and Surgical Journal.
 Bristol Medico-Chirurgical Journal.

British and Colonial Druggist.
 British Journal of Dermatology.
 British Medical Journal.
 Climate.
 Clinical Journal.
 Clinical Review.
 Giornale Medico del R. Esercito.
 Il Policlinico.
 Indian Engineering.
 Indian Medical Gazette.
 Indian Medical Record.
 Janus.
 Journal of Balneology and Climatology.
 Journal of Laryngology and Otology.
 La Grèce Médicale.
 Lancet.
 Liverpool Medico-Chirurgical Journal.
 Medical Brief.
 Medical Missionary Journal.
 Medical Record.
 Merck's Archives.
 New York Medical Journal.
 Pacific Medical Journal.
 Polyclinic.
 Public Health.
 Revista Medica de S. Paulo.
 South African Medical Journal.
 The Hospital.
 The Medical and Surgical Review of Reviews.
 The Northumberland and Durham Medical Journal.
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- 1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.
- 2.—Manuscripts sent in cannot be returned.
- 3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.
- 6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

ON TROPICAL ANÆMIA, AND ITS RELATIONS TO THE LATENT AND TO THE MANIFEST FORMS OF MALARIAL INFECTION.¹

By ALBERT PLEHN, M.D.

Physician of the Imperial Government in Cameroon.

(Continued from p. 123).

THE general opinion seems still to be that, between the single fever attacks as well as during the incubation stage before the first attack, the malaria plasmodia develop and increase in the well-known form continually in the body of the infected individual, and further, that this process goes on until they are numerous enough to bring on a fresh attack of fever. Koch and Ziemann also take this for granted. The former calculates, from the period of incubation of ten to fourteen days, "that five to seven generations must have developed before the disease proper can break out."²

Now it is not quite clear why the fourth or sixth generation, for example, should not bring on at least a slight fever, if, as is widely supposed, the parasites increase during the incubation period through sporulation. Even if it be possible to explain (as the Italian observers do) the well-known lack of sporulation forms in the peripheric circulation, through the fact that the

mature parasites are held back in the capillary system of the inner organs, it is, however, not clear why the young amœba-like forms originating from the first to the sixth generation should avoid the periphery, whereas those of the seventh generation are suddenly present in great quantities in the peripheric blood.

And yet it is a fact that, up to four or five days before the attack, not a single parasite is to be detected; on the third day, perhaps, a few extremely minute forms may be found. On the contrary, the "primitive forms" are present, and, if lucky, the observer may discover, usually on the second day before the fever attack, the above-mentioned transition forms, the karyochromatophilic granules being modified to minute plasmodia. These, after about forty hours, are fully grown, and bring on, through their sporulation, an attack. I say, "if lucky," for the metamorphosis and the first growth of the young parasite seem to take place pretty quickly, so that only for a few hours are the really convincing forms to be seen. If the usual double attack be now stopped through two good doses of quinine, only the younger forms survive in the blood, and these develop, as described, in four to six days to maturity. Then may again follow the metamorphosis to plasmodia, which after forty or forty-eight more hours reach the sporulation stage, thus bringing up the total interval to about seven days. Fisch reports that the eight-day fevers are frequent on the Gold Coast in the case of patients who are going to have blackwater fever, and such fevers occur also in Cameroon, even if less frequently. Usually in Cameroon the change of alternate generation does not occur already after five days, since the propagation takes place again through direct division, and it is only after the second generation has matured that the metamorphosis follows. This would give $2 \times 5 + 2 = 12$ days, and reckoning, as is usual,

¹ Paper read before the Berlin Medical Society, May 31, 1899.

² According to this point of view, the number of the preceding sporulations must have been still greater in the cases which have been carefully observed, when the first fever attack occurred many weeks after the possibility of infection. This remark also applies to cases in which relapses followed many months after the individuals had left a fever region, and after they had been without fever for a long period.

from the first fever attack, we get exactly the fortnightly type which is characteristic for most tropical fevers.

I must confess, gentlemen, that these conditions are by no means always so evident, for the primitive forms are generally of different sizes. Their growth may be clearly followed, especially after blackwater fever, for in this case all parasitic bodies, down to the youngest germs, are, as a rule, destroyed with their hosts, the red blood-cells.

The typical incubation period of twelve days, observed in cases where the possibility of infection was limited to hours, may be explained in the same way as the interval between two attacks. The *apparent* incubation period, according to reliable observations, of only forty-eight hours, and the *apparent* rapidity with which single injurious causes act, such as colds, getting wet, excesses or sudden change of climate, depend upon the fact that at the time of injury a sufficient number of parasitic primitive forms, always present in the blood of infected individuals, happened to be mature and underwent the modification to plasmodia. I do not know of a single positive case in which the illness followed the injury within less than forty-eight hours, and in which parasitic development could be proved.

It seems to me that the action of the primitive forms of the parasite offers likewise a plausible explanation of the aetiology and the course of blackwater fever.

The explanation of this disease which I offered three years ago pointed out that the acute decomposition of the red blood-cells, which characterises it, is principally due to an especially intense or prolonged action on the organism of the malaria poison, whether this action bring on numerous acute attacks or not. Thus originates the *predisposition*.

For the *outbreak* of the paroxysm the favourable moment is necessary. In the very great majority of cases this may be a simple malaria fever as soon as it is treated with quinine. This view has been but strengthened through the experience I have gathered since then, and it agrees pretty closely with the more recent explanations of Bignami and Bastianelli. Moreover, it is extremely rare that a malaria fever *without quinine treatment* is complicated with hæmoglobinuria, and still more rarely does quinine *alone* cause the blackwater fever in predisposed individuals without the co-operation of the malaria parasite. That the former does not occur oftener in Cameroon is due, without doubt, to the fact that luckily there it is only in very few cases that a quinine treatment is not employed for malaria. The second occurrence represents the maximum of blood decomposition. In whatever way, however, the blackwater fever may be called forth, the clinical course in about 100 cases which until now I personally investigated was quite independent of the cause, and the cure followed, if at all, so regularly without the use of quinine (*also when quinine had not been administered before the outbreak*) that I have no occasion to divide, with Bastianelli, the blackwater fever in different groups.

And now, gentlemen, allow me a digression. From my description of the part played by quinine occasionally in the aetiology of blackwater fever one might be led to discredit quinine as an anti-malaric agent. Indeed, similar statements made by one person

of greatest authority have had this effect in certain quarters, a result very much to be regretted, and which could hardly have been the author's intention. I therefore consider it my duty to express most emphatically, just at this point, that quinine, or euquinine respectively, remain to this day, in spite of the occasional action in inducing blackwater fever, as the only specific we possess for malaria. Its use cannot be recommended enough, at least in Cameroon, in those cases in which spontaneous healing by blood dissolution and hæmoglobinuria has not yet set in.

Of the few simple malaria cases in Cameroon, in which owing to various reasons the quinine treatment had, exceptionally, been employed in insufficient doses, or not at all, *eight* ended fatally only within the last two years. In five cases the fever occurred for the first time; in five others the illness lasted two to four days; in one case eight, and in one twelve days. In the eighth case the length of the illness was not certain. Quite recently a ninth case occurred in the person of an English colleague from Akkra, who, after a stay of six weeks on the Coast, embarked on a healthy state for England, was taken ill shortly after with malaria, and died on the third day before a quinine treatment could be decided upon.

A tenth case under similar conditions occurred recently in East Africa.

It is, therefore, easily understood that I did not care to test the possibility of spontaneous healing in the case of the Cameroon malaria. In cases in which I had reason to fear the outbreak of an exceptionally severe blackwater fever after the use of quinine, I have often put off from day to day the prescribing of the "blood poison." In the end, however, with but one exception, I had always to resort to the use of quinine, which naturally at a later stage acts under conditions still more unfavourable.

The practitioner in the tropics must consequently realise the responsibility he assumes in not adopting immediately a quinine treatment, either because he fears an outbreak of blackwater fever, or because he counts on a spontaneous cure, or owing to some other reason.

After the outbreak of hæmoglobinuria, however, the conditions are quite altered. It was Friedrich Plehn who first conclusively pointed out in Germany the extraordinary tendency which blackwater fever has of healing spontaneously, and who counter-indicated the use of quinine in such a case. I, myself, was then able to offer an explanation of the process of healing, viz., that the plasmodia are destroyed in the plasma owing to the dissolution of the infected blood-cells, their hosts. It is consequently quite natural that Geheimrath Koch almost never found them, and I consider he has gone a little too far when he therefore concludes that "blackwater fever has directly nothing to do with malaria."

Leaving out of consideration that, except in quite individual and extremely rare cases, numerous malaria parasites are almost always to be found in the blood *before its dissolution begins*, how can an *intermittent blackwater fever* be accounted for through quinine poisoning alone? Or seeing that cases have really been observed by me and by other authors in which the previous use of any quinine could be excluded

with certainty? Or how explain the cases in which quinine had been administered for many days before?

Moreover, if the action be due solely to poisoning, how is it to be explained that a malaria patient, who one day gets a severe blackwater fever from 1 to 1½ grams quinine, can support 6 to 8 grams a few days afterwards without getting hæmoglobinuria—an observation which induced Steudel to try such excessive doses as a remedy for hæmoglobinuric malaria.

The *nature* and the causative conditions of the reduced resistance of the erythrocytes are up to this moment a complete mystery, although sometimes it can be proved to exist even experimentally. I am convinced that the *modification of the blood's constitution is a result, even if indirectly, of the development of the parasitic germs.*

As late as three years ago, I was compelled through the experience I had gathered to look upon the well-known form of the malaria-plasmodium as a necessary factor in the development of blackwater fever. Since then, I have had the occasion to observe (as Murri and Bastianelli have done) that blood-dissolution can *exceptionally* take place without the presence of plasmodia. At the time I was unacquainted with the germs, but even they are hardly ever numerous enough to allow one to explain the hæmoglobin reduction through the decomposition of the infected blood-cells alone. It is true that, at least in Cameroon, the latter are the first to decompose, but when the amount of hæmoglobin is reduced in a few days by 40-50 per cent., the supposition necessarily gains that the process of dissolution has also spread to blood cells apparently unchanged, morphologically. This view is further supported by the fact that the dissolution often continues even after the infected blood disks have already disappeared from the circulation. *Well, then, I consider the diminished resistance of the apparently normal blood disks as a consequence of the fact that the blood-building organs are at last incapable of regenerating enough blood to meet the unusual and persistent demand brought about by the continual dissolution, and finally yield in part a deficient product.*

A further observation supports this explanation, viz., that even an excess of plasmodia and germs during the first months of a stay in Africa never lead up directly to blackwater fever, even if a great deal of quinine be taken, whereas individuals severely infected run every chance, at a later period, of falling victims to it. This view also explains how, when once the deficient blood disks have been expelled through a severe attack of blackwater fever, a convalescent can often stand a strong dose of quinine, as proved especially by Steudel's and Berenger-Ferraud's accounts of cases. The truth here is that the elements susceptible of being poisoned are lacking. Only a few weeks after the attack are these elements sufficiently regenerated in order to start a new blackwater fever through their decomposition, on the malaria parasites cooperating with the action of the quinine, for, as already mentioned, the blackwater fever results only exceptionally through the influence of one of these two factors. In short, the predisposition remains until the blood-building organs are again capable of delivering only a perfect product, inasmuch as the excessive demand on their activity has been reduced.

This can speedily follow when, for example, during a very severe blackwater fever also the germs of the *chronic* infection have been destroyed, and through some reason or other a new infection does not take place for some time. This rapid disappearance of the predisposition which occasionally occurs does not seem to me to be easily accounted for through Bignami's and Bastianelli's theory, which ascribes to a specific change in the blood-building organs the diminished resistance of the product they yield. In any case, it is a fact that the capacity for regenerating blood may continue *quantitatively* to the end. When, notwithstanding, the number of blood corpuscles and the hæmoglobin remain considerably below the normal in individuals infected with chronic malaria, the reason is, probably, that man possesses normally an excess of organs for internal respiration which he can do without at times of active dissolution, in which case then the absolute need may be partly supplied without the possibility of any excess being formed. The very few observations carried out up to date in other malarial countries—for example, in Italy—agree with my theory.

In the latter country I found the karyochromatophilic granulation in ten out of eleven inhabitants of the rural districts of Macarese, which is one of the worst fever regions of the Roman Campagna. In these individuals, excepting in one case, the karyochromatophilic granules were very scarce and sparsely developed. No wonder, then, that the phenomenon escaped hitherto the Italian observers, or was misconstrued. Only through the studies on the alterations of the blood previously carried out in Cameroon, where they have quite another importance, was I able to detect them also in Italy.

The usually weak and limited development of the "primitive forms" of the parasite in Italy explains why in that country the blackwater fevers are so extremely rare, although the use of quinine is general in the country, and it is estimated that 15,000 deaths are due to malaria every year.

Germs of parasites acquired in other parts of the world were quite similar. However, in some of the latter cases the occasion when infection had been possible lay far back, and it would not be safe to conclude that the same conditions hold in the seat of infection from the rare granulation found in the observed individuals at Hamburg.

My theory seems to a certain extent to contradict the physiological experience that young cells are usually especially resistant. Nevertheless an extremely active blood regeneration does without doubt take place. This is proved in the case of anæmic Cameroonians by the nucleated erythrocytes almost always present in greater or smaller numbers. Besides, in more serious cases there are also poikilocytes and even megalocytes. Moreover the great increase of hæmoglobin is also directly noticeable if, under certain conditions, the destructive action of the parasitic primitive forms suddenly stop; one may then often notice a daily increase of several units per cent. in the amount of blood.

The fact that, after a prolonged stay in a fever district, continually decreasing doses of quinine suffice to arrest the individual attacks is probably connected

with the gradually diminishing resistance of the red blood disks, brought about by the chronic malaria infection; nay, in individuals with a predisposition for blackwater fever a simple malaria attack may occasionally heal spontaneously, even in the fever locality itself. For precisely in such cases a minimal dose of the blood poison, or even the parasites' invasion alone, suffices to destroy the infected blood-cells, and with them their guests. In the beginning greater doses are necessary to accomplish this object, and it is this process which, properly speaking, constitutes "a cure" in most cases.

The systematic and prophylactic use of quinine, if prolonged, affords a real protection not only against acute fever attacks, *but also* against the excessive growth of the primitive forms, and therewith against the blackwater fever. I shall report on this point in a special work. The action of quinine also supports the view that there is a relation between the bodies we have described and the malarial infection.

However, even if some of the details of the explanation which I have evolved should have to be modified, the connection of the karyochromatophilic granulation with the malaria infection may well be considered proven as far as West Africa is concerned. Considering the concordant results of the few investigations I was able to carry out in this direction, I have no doubt that the same relation will soon be proved to exist in other malaria regions also.

The practical benefit derived from the knowledge of the germ of the malaria parasite accruing first to West Africa would therewith become general.

The expert will now be in a position, by examining the blood every month or two, of recognising when an extensive development of karyochromatophilic granules demands urgently the prophylactic use of quinine, which was perhaps deemed unnecessary at first. He will further be able to determine when settlers, of whose case it is impossible to stay the proliferation in the primitive forms, will require leave of absence to avoid the risk of blackwater fever, and whether an early relapse is to be feared, after the blackwater fever has been successfully overcome, owing to extensive granulation having occurred in the blood disks during convalescence, a fact which would justify leave of absence.

It is of special importance that in the future every physician who is acquainted with the karyochromatophilic granulation will be capable, even if he have no experience of the Tropics, to determine objectively when the malarial infection has disappeared, and therefore to decide when the individual on leave may return to the fever district *without* running the danger of getting blackwater fever. This danger is not unfounded, *for the infection may for want of proper treatment have continued to spread during the absence*, and blackwater fever might then follow as a result. Unfortunately this happened several times in Cameroon.

Naturally the disappearance from the blood of the germs of infection is not enough to warrant the individual's return; he must *hereafter* allow the blood-building organs time to recuperate. The length of this period should depend, of course, from the degree of exhaustion, and this will generally coincide, more

or less, with the time the infection lasted. In special cases digressions from this rule should be justified by investigating the blood in the manner described above. However, the conditions described explain the experience made of old in Cameroon, that a short leave to districts free from malaria has no noticeable influence on the time within which the first blackwater fever outbreak follows, even if the usually nervous inhabitant of the Tropics feel subjectively very much better for the change.

We can further determine objectively *whether a district be malarious or not* from the presence or absence of karyochromatophilic granulation in the blood of the natives. It is true that for the present this only applies to the West African coast, but it will soon be possible to make this general. In such an investigation one will have to exclude very carefully an infection which has taken place in a neighbouring malaria district. As pointed out already, the germs may be acquired, and may multiply in the body for months and years, without causing any other symptom but a slight anæmia, which can only be determined through the use of the hæmometer. During this incubation period, moreover, attacks of fever may be altogether absent. In two cases I was able to detect not only the "primitive forms," but also the plasmodia developed therefrom, two years after any chance for infection had been possible, and this, although the subjects had been, and were at the time of the examination, quite well.

The analogy between the malaria of man and that of cattle (Texas fever) holds also in this regard, and Koch has recently again called attention to it. Smith, the discoverer of the Texas fever parasite, found that the blood of "healthy" cattle from infected regions proved infectious for cattle from districts where the plague was absent, even after a period of three years.

Members of the Navy may possibly grow very anæmic owing to the development of the malaria germs, and may leave Cameroon *without ever* having had fever. They may then often be taken ill with malaria on the journey home, or upon reaching Europe, although it is possible that they remain altogether immune.

I remember two cases which occurred during my assistantship at the Friedrichshain Hospital that prove the extraordinary duration of latent infection. My highly esteemed teacher, and at the time director, Mr. Fürbringer, informed me that these cases are not the only ones known. Once after typhus abdominalis, and once after pneumonia, a fever broke out with the characteristics of an intermittens malarica, which rapidly disappeared on using quinine. It was impossible, through the most careful questioning, to elicit any information as to whether the patients had ever had an intermittent fever, or had ever lived in malaria districts. The infection must have been extremely old,

I only had the opportunity of getting acquainted with the most recent and very interesting investigations of Ross, of Koch, and of the Roman observers, Bastianelli, Bignami, Dionisi and Grassi, as I was on the point of leaving Africa and as I got to Rome. The above work had then been already concluded but

for the comparative observations carried out in Europe. As is well known, the latter investigators have succeeded in several instances in following in the stomach and in the abdominal cavity of certain kinds of mosquitoes the development of certain forms of the malaria parasite of man in the Roman Campagna. They have also, once successfully, inoculated the disease to a healthy individual through the sting of an infected mosquito. It remains still to be seen if their results contradict my observations, and to what extent.

In the drawings of Sporidia from the salivary gland of mosquitoes which Grassi showed me in Rome, there certainly was a certain resemblance between the chromatin (stained with hæmatoxylin) and the karyochromatophilic granules. It would be unsafe to conclude on their being identical owing to this resemblance, since, as far as I know, zoologists do not ascribe to Sporidia a process of propagation through direct division lasting months and years; whereas, owing to the clinical observations, one is forced to accept this explanation in the case of malaria germs. I have, therefore, avoided (as might seem natural) to describe the karyochromatophilic granules as the "spores" of the malaria plasmodia. Further, I considered that the expression "enduring forms," although in a manner it expresses some of the salient properties of the parasites, would be as unsuitable, as it carries with it an idea of rest. I was, therefore, led to speak of "primitive forms," admitting to myself at the time that also this name can only be justified, to begin with, in the case of West Africa, and that to many it will seem antiquated.

My observations in Cameroon certainly contradict the theory, at least as taught up to now, that the *only* manner of infection is the direct transmission through certain kinds of mosquitoes. I do not lay so much stress on the fact that in the Jossplatte, the most dreaded fever locality in our colony, mosquitoes of any kind, as well as other stinging insects, are extremely rare, and also that in other places, as for example, Kribi, they are said to be altogether absent. My brother and Ziemann have already pointed this out. If, as the Roman investigators maintain, a single mosquito sting (which in the disease-transmitting species is supposed not to be even felt) is enough to bring about an infection, then the possibility of this is nowhere to be excluded, not even through the use of mosquito-nets. In Cameroon, not the slightest effect was noticed, whether the latter were used or not, on the term of the first fever attack, or on the number of relapses later on. It is much more important to notice that the half-moon forms (crescents) of the malaria parasite, which alone are supposed to be capable of transmission, are in Cameroon extremely rare; in fact, they occur quite isolated, and are not to be found during many of the worst fever months.

I saw during almost four years in Cameroon but once flagellated forms. Perhaps it is that the primitive forms, which are always present and also to be found almost in all natives, undertake their part.

These considerations, however, are not offered to throw doubt on positive success, but as a warning against the unlimited generalisation of local observations which seems to be in vogue to-day, and which

is not conducive to gaining a clear insight in the question of malaria.

The next part of the problem is to determine clearly the possibility of transmitting a real infection of "primitive forms," and so determine their geographical diffusion. The realisation of the first task was impracticable in Cameroon, because it was never possible to exclude with certainty that a previous infection did not already exist. I have taken up, as far as this is possible in Germany, the investigation of the second part of the problem.

A CASE OF GOUNDOU OR ANAKHRE.

By Dr. W. RENNER, Sierra Leone.

(See Plate in this issue.)

S. L., a fisherman, aged 39. Grandfather of the Jola tribe, grandmother an Ijesha of the Aku tribe, both liberated slaves settled in the village of Kissy, where his father and himself were born. On his mother's side he comes from the Egba of the Aku tribe.

He has lived all his lifetime in this village with his brother. He is married and has four children; wife has never miscarried, has no trace of syphilitic disease. He has never left this colony, and has always resided in his native village, and has enjoyed comparatively good health.

He was not born with any fulness of any part of the face, and those who knew him from childhood have never noticed the swelling about the nose until within recent years.

He gives the following account of his illness. He has suffered from occasional cold in the head, but it was not until 1882 that a small fulness was noticed on either side of the nose; this has been growing, and now has the appearance of half a pigeon's egg, lying obliquely along the nasal process of the superior maxillary bone. With the growth of these eminences there have been occasional persistent headache and sanguino-purulent discharge from the nostrils. There is no pain on pressure over these eminences, which are hard and smooth, and the skin is not adherent to the subjacent bone. The cartilage of the nose and the nasal duct are not involved. The hard palate is not affected, although he speaks with certain amount of nasal accent. In other respects this man is strong, and pursues his daily vocations with only an occasional interruption due to chronic rheumatism.

Remarks.—This case supports Lamprey's observation as to the wide distribution of this rare and peculiar disease, and that it is not confined to any particular tribe of Africa.

The symptoms in this man's case agree with those described by Dr. Macleod and quoted by Dr. Manson in his work on Tropical Diseases. As to the etiology of the disease nothing is known, and Dr. Macleod's hypothesis as to the disease process being started by the larvæ of some insect could not be entertained. This man has never left his home; cases of this disease have not been seen in the village, which is situated on the right bank of the Rokelle or Sierra Leone River.

TREATMENT OF A LEPER BY SUBCUTANEOUS DOSES OF CHAULMOOGRA OIL.

By Dr. TOURTOULIS BEY.

Communicated by F. M. Sandwith, M.D., M.R.C.P., Professor of Medicine, Kasr-el-Ainy Hospital and School, Cairo.

A COPTIC Egyptian living in Upper Egypt was attacked by nodular and anæsthetic leprosy in 1879, when he was 15 years old. He says that there have been cases of leprosy in his village, but never in his family. Five years later, during one of the temporary lulls of the disease, he thought he was cured and then married.

He was treated by various European doctors in many ways until 1889, when he first consulted Dr. Tourtoulis, an Albanian medical man, who was educated in France. During that year the former of these two photographs was taken, showing well an advanced case of leprosy of ten years' growth. The many nodules on the face, ears, lips, nose and

were beginning to break down, his breath was stinking and the soft palate was ulcerating, while his fingers had become atrophied and claw-like. There were, in addition, two deep perforating ulcers on the sole of the right foot and a wound on the dorsum of the left foot. Also his sight had become defective; there was evidence of double broncho-pneumonia and a spasmodic laryngeal cough. The patient refused to take more chaulmoogra by the mouth, so Dr. Tourtoulis decided in May, 1894, to give him some subcutaneous injections of the oil in doses of 5 grammes. After fifty injections, to his great surprise, he saw an improvement in the general condition, and the complete disappearance of the ulcers on the feet.

At the end of August hairs began to grow on eyebrows and chin, and the sweat glands, which had been quite inactive, began to secrete on the face and limbs.

In September the patient could walk, and went to his country home to look after his cotton crop. Early in 1895, besides a gradual continuance of improve-



chin, with an absence of eyebrows and moustache, contributed to the well known leonine aspect. There were, in addition, a sore on one finger, enlargement of ulnar nerves and much anæsthesia.

For five years he was treated off and on with tepid soda baths, nasal antiseptic douches and chaulmoogra oil internally, varying from 30 to 50 drops three times a day in a glass of milk. Repeatedly the oil had to be stopped, because it invariably produced nausea, vomiting and loss of appetite. The patient returned in despair to his village until February, 1894, when he re-appeared in a worse state than ever. His face was covered with leprosy nodules, some of which



ment, it was noted that there were no longer any sores, the facial expression had changed for the better, and the anæsthesia had partly disappeared, so that the hypodermic injections had become painful. But the hands and fingers remained claw-like, though they enabled the patient to begin writing again. The injections were continued at intervals till May, 1899, in spite of the objections of the patient, who considered himself cured, for at least a year. During the five years some 544 injections were made, amounting to 2,750 grammes, or about 96 ounces of chaulmoogra oil.

The injections were invariably made on the outer side of the arms or legs with a long needle, and strict

antiseptic precautions. The best chaulmoogra oil was obtained from Paris in sterilised tubes.

Remarks by Dr. Sandwith.—I was invited to see this patient by Dr. Tourtoulis for the first time in April, 1899, and I was so much struck with the obvious improvement in his condition that I suggested a second photograph in the same attitude as the first, for comparison. This has been done with some success. Anyone well acquainted with leprosy would easily have recognised the disease last April on examining the man's face, ears, ulnar nerves and hands. The fingers were still somewhat contracted and the inter ossei slightly atrophied. On the other hand the face had lost its pigmented nodules and had regained a fair share of eyebrows, eyelashes and moustache.

The writing powers of the patient can be best referred to by saying that he has lately been made the chief representative in his province of the New National Bank. From being a social outcast because of his disease, he has now become an European Vice-Consul. He is a remarkable man, possessed of energy and perseverance which is quite rare among the inhabitants of Egypt.

Some of my English friends have known him for many years as an extra intelligent member of a respectable family of farmers, whose commercial honesty is beyond question. One friend also assures me that some years ago the patient was one of the handsomest natives of Upper Egypt. His social position is worthy of note, because all the other male lepers I have seen in this country have belonged to the beggar class, or at best have been labourers working for a daily wage. Chaulmoogra oil, as an internal and an external remedy, has been well known to the profession since 1879, and during the last few years I have often tried it on lepers, but though I have met with some cases of temporary improvement I have never seen anything like the good result achieved in this case by Dr. Tourtoulis's indefatigable perseverance. I consider the case worth reporting in this Journal, because it is encouraging to meet with any cessation of symptoms in a so-called incurable disease, and because it may incite others to try this remedy upon early cases, and publish the results.

I have at present under treatment a young girl who is known to have been leprosy for at least six months. She has many well marked nodules, several maculæ and anæsthesia of feet. Injections of the chaulmoogra oil in her case produce slight pain for two or three hours, and a local swelling for two days, but no rise of temperature. Following Dr. Tourtoulis' advice, the patient is also having daily warm baths, each containing 200 grammes of carbonate of soda. I must not forget to mention that Dr. Hallopeau and Dr. Jeanselme in Paris a year ago injected several lepers during some weeks, relinquished the treatment, and have now taken it up again in consequence of the report of this case in the *Bulletin de la Société de Dermatologie*, July 13, 1899.

In conclusion, I must say that I do not regard this Copt as cured of leprosy. On the contrary, I think he is still a leper, with a very unusual improvement in his condition. Also I do not forget the rare possibility of a complete permanent spontaneous arrest of the disease, as exemplified a few years ago

by a female patient shown by Mr. Jonathan Hutchinson at the Medico-Chirurgical Society. She was then perfectly well, but she had been the subject of undoubted tubercular leprosy twenty years before. In her case, if I remember rightly, no special treatment had led to this result.

LIGHTNING STROKE.

By R. E. ADAMSON, M.B., C.M.
Labuan, North Borneo.

On the evening of August 13 there were indications of heavy clouds coming up from the south-west towards the Island of Labuan, and shortly afterwards a severe storm, with heavy rain and high wind, commenced. At its height there were four distinct and exceptionally vivid flashes of lightning, accompanied by simultaneous deafening peals of thunder apparently just overhead. One of the flashes struck a house with its occupant, and the following notes may be of interest:—

(a) INJURIES TO OCCUPANT. (b) DAMAGE TO HOUSE AND ARTICLES.

(a) INJURIES TO OCCUPANT.

The subject of this paper was seated at dinner, and at the moment the lightning struck the house he felt a terrific blow on the left knee as though it were caused by something solid and round. He fell off his chair unconscious, and remembered nothing more until awakening up from his unconscious state to find himself unable to get up and in total darkness, with the appearance of burning specks on the floor around him. The Chinese "boy," who was present in the room, ran away, and it was some little time before assistance could be obtained to lift the patient into bed. The legs showed the following condition:—Both legs were swollen and green in appearance, this latter passing off in a short time. From the knee to the toes of the left leg the skin in some places was torn away; in others it was raised into blisters. From middle of thigh to ankle of right leg the condition was the same as in the left leg. The patient states that he felt as though he had no legs at all, being quite unable to appreciate any movement at the ankles or toes. Sensation was completely lost, and the feet felt very cold. There was great pain in the muscles of the legs. General shock was considerable, and deafness with ringing sounds in the ear.

The patient made a rapid recovery after the application of hot-water bottles and massage to the soles of the feet and emollient dressings to the burns. At first he was unable to put his feet to the ground, but after one week could move about slowly, and now only feels a peculiar pricking sensation in the big toe of the left foot when the skin is scratched or rubbed over the instep.

(b) DAMAGE TO HOUSE AND ARTICLES.

The House has an attap roof with kadjang walls, and is raised on wooden piles. The dining-room is central, and the dining-table occupied the middle of the room, above which a large brass lamp is suspended

by thick wire. When the flash came, it struck the top corner of the roof, ran down one of the principal supporting posts, splintering it very badly, along the main beam to the wire support of the lamp; travelling down this, it made a jump to the fruit stand on the table; thence on, encountering cruet, knives, spoons, forks, &c., making a hole in the table; thence by way of the table cloth, clothing and legs of patient to the floor, and passing through this to the supporting piles, splintering two or three of them into matchwood, and then finally to earth.

Lamps.—The shade of the lamp was broken, and a brass plate fixed on by thumb-screws to the bottom of the oil reservoir was thrown to the other side of the room.

Fruit-stand.—Consisting of two mother-of-pearl shells, silver mounted. Handle fused and discoloured; other portions slightly discoloured.

Large Knife.—Point fused completely off, with blue discolouration for about an inch. Large blue patch where handle joins the blade. Blade also fused at this point; handle slightly charred.

Small Knife.—Point slightly fused, with about half-inch of bluish discolouration. Where blade joins handle, the edge and back of blade fused; handle discoloured.

Butter-knife.—Silver; point and end of handle badly fused.

Cruet-stand.—Silver salt-cellar with silver supporting band-cellar fused into stand at two places; band fused off the stand; other places slightly discoloured. Salt-spoon handle partly fused, bowl of spoon badly fused.

Dessert-spoon.—Handle fused, end of spoon badly fused and discoloured.

Table-cloth.—The part at which the patient was sitting was torn to ribbons, with large piece missing. At only one spot could a trace of charring at the ragged edges be found.

Table.—As nearly as possible under the patient's plate a hole was made, with the surrounding parts discoloured as if by explosion.

Clothing.—Some of the patient's wearing apparel was torn into shreds.

Shoes.—Of a native pattern with uppers ornamented with beads and gold and silver threads. The left shoe had its threads fused and discoloured, and wherever there was a nail in the shoe there was a hole burned in the leather, and the nail partly fused.

The patient has the habit when wearing shoes of this pattern of frequently sitting with one foot out of the shoe, thus explaining why the right foot was only burned to the ankle, and the shoe free from all discolouration and fusing.

Wooden Piles supporting House.—Two or three piles supporting the floor immediately under the table were splintered into fragments, uprooted and scattered round the holes they had previously occupied.

In addition to the above enumerated damages there were many others, including the bursting and breaking of several bottles containing aerated waters. A weekly paper which the patient was reading at the time was partly blown out of his hand. It is also interesting to note that the plate which the servant was about to place in front of the patient was broken

in his hand, and he (servant) was in no way hurt beyond receiving a shock and great fright.

I record this case, hoping that it may be of interest not only to practitioners abroad but also to medical men at home, as one reads so little of lightning stroke, and most, if not all, of the treatises on medicine omit the subject, or only treat of it lightly.

The course of the lightning was unmistakable; the almost complete absence of charring to clothing and the absence of after-effects are points worthy of notice.

Some two or three years ago I had occasion to examine three Malay men who were killed outright by lightning in a boat, but no *post-mortem* examinations were granted to enable one to ascertain the state of the internal organs, should there be anything peculiar in such a condition.

TUMBE, OR KROO FLY.

By A. H. HANLEY, F.R.C.S.I.

Niger Coast Protectorate.

THE history of above-named fly, from the time its egg is deposited in or on the skin, may be of interest. On June 7 last I saw at one of my out-stations a European who complained of a boil that would not heal. On examination I detected a larva in it, which I removed and placed in gin intending to examine it on my return home. After it had been in gin for at least twenty minutes the bottle was accidentally broken. As the larva, notwithstanding the length of time it had been in the gin, was still alive, I put it into some cotton wool, and on my return home two hours afterwards I placed it in a bottle containing hot boiled rice. It moved about the bottle for three days and then entered chrysalis stage. Fourteen days after entering chrysalis stage a fully-formed fly appeared. The patient says boil began to form fourteen days before he consulted me. Allowing that the irritation commenced within twenty-four to forty-eight hours of the egg being deposited, the interval that elapsed between deposit of egg and appearance of fly would be, roughly, one month. These flies somewhat resemble a horse-fly, and are common in parts of the Niger Protectorate. Mr. Austin, of South Kensington Museum, was kind enough to show me several specimens of this fly which he had collected during his recent visit to Sierra Leone.

THE honour of C.M.G. has been conferred on Patrick Manson, M.D., Medical Adviser to the Colonial Office, John Pringle, M.B., Member of the Privy Council and Legislative Council of the island of Jamaica, Wordsworth Poole, M.B., for services as Principal Medical Officer of the West African Frontier House on the Niger, and Dr. A. Mackinnon, Principal Transport Officer for Uganda, for services in connection with the recent Uganda Mutiny.

ON THE METAMORPHOSIS OF THE YOUNG FORM OF *FILARIA BANCROFTI*, COBB [*FILARIA SANGUINIS HOMINIS*, LEWIS; *FILARIA NOCTURNA*, MANSON] IN THE BODY OF *CULEX CILIARIS*, LINN., THE "HOUSE MOSQUITO" OF AUSTRALIA.

By THOS. L. BANCROFT, M.B. EDIN.

(Read before the Royal Society of N. S. Wales, June 7, 1899, and reprinted from the Journal and Proceedings of the Society.)

DR. PATRICK MANSON, in a paper read before the Linnean Society of London, March 6, 1884,¹ remarked:—"Six years ago I described the metamorphosis undergone by the embryo *Filaria sanguinis hominis*, in the body of the mosquito.² I hoped that (considering the practical importance of a correct knowledge of the life-history of this parasite) the statements I then made would, long ere this time, have been thoroughly confuted or confirmed. . . . With the exception of Lewis in India, Myers in Formosa, and Sonsino in Egypt, I do not know that anyone has worked seriously at the subject. And although both Lewis and Sonsino have confirmed my statements as to the entrance of the *Filaria* into the mosquito, and followed up part of the metamorphosis, neither of them has advanced his observations so far as to be able to confirm my statements as to the later stages of this, or positively to prove that the mosquito is, or is not, the intermediary host. Some eminent helminthologists in England accept my statements and endorse the inferences I have drawn—Cobbold, for example. But in other quarters, so far from securing acceptance of my theory, the work of Lewis, on account of the hesitation and scientific caution with which he expresses himself, has had the effect of inducing a certain amount of scepticism. Leuckart is sceptical; and, of course, the scepticism of so eminent an authority is of great weight in influencing opinion, especially in Germany."

In answer to an inquiry from me as to whether there was any recent work on the subject of "filarial metamorphosis," Dr. Manson wrote, November 15, 1898:—"So far as I know, nothing has been done in 'filarial metamorphosis' since my Linnean Society's paper. Lewis did not go very far with the work. There is an excellent opportunity for work on this subject, and were I in your place, I should certainly go on with it."

In writing to Dr. Manson, I had mentioned the circumstance of my being able to verify his "filarial metamorphosis, but that I had never seen the "actively moving filaria," which he stated left the mosquito's body and lived a free life in water until transferred to the human host.

To this he replied in these words:—"I have seen the 'actively moving filaria' in the seven days old mosquito a good many times; I used to be able to pick out the mosquitoes containing it. Their thoraces looked plump and juicy to the eye. Of course you must have hundreds of mosquitoes from which to select such."

Now, in my former investigation, there was no difficulty in finding the early stages of the metamorphosis in every mosquito (*Culex ciliaris*) without exception, which had imbibed filariated blood; those mosquitoes which lived seven days—and none ever lived longer—never contained any actively moving filariæ.

It were useless to make further search for this "actively moving filaria"; either Manson must be in error, I thought, or the *Culex ciliaris* was not an efficient host.

The recent work in India on the metamorphosis of the malarial parasite in mosquitoes induced me to study the habits of these insects in this district. I found that I could keep certain kinds of mosquito, particularly *Culex ciliaris* and a large black species hitherto undescribed, alive in confinement for about two months; one individual actually lived seventy days. Banana was found to be a good food for them. It was ascertained that unimpregnated mosquitoes lived the longest; those that had been impregnated lived two or three weeks, whilst the males rarely lived a fortnight. In my former investigation into filarial metamorphosis, it never occurred to me to feed my filariated mosquitoes whilst in confinement, accepting the common belief of their only feeding once, and dying within a week. Manson, Lewis, and Myers, who had worked at the subject, never fed the mosquitoes, and it never dawned upon me that my mosquitoes were dying from starvation.

Having discovered that mosquitoes could be kept alive long periods in confinement if fed on banana, I was anxious to ascertain what would become of the filariæ, which were to be seen in mosquitoes that lived seven days; would they go on developing if the mosquitoes lived longer?

Unfortunately, E. S., the filariated subject, a girl of 16, from whom I had obtained filariæ, had left the district, having secured a situation as domestic servant; she was the only person affected with filariasis I knew of.

Dr. Manson's encouragement and a grant of £7 from the Queensland Branch of the British Medical Association, to defray the cost of E. S. returning and living with her parents for three months and submitting to be bitten by mosquitoes, induced me to enter upon a fresh investigation on February 1, 1899. It was found that the actively moving filariæ were to be seen, but not before the sixteenth or seventeenth day, sometimes in cold weather not until twenty days, and that no further development occurred in them even after a sojourn of sixty days in the mosquito's thorax.

The final stage of the metamorphosis, i.e., the preliminary alternation of generations, is attained in sixteen or seventeen days; the young filariæ are then $\frac{1}{16}$ in. in length by $\frac{1}{160}$ in. in breadth, some only $\frac{1}{16}$ in. by $\frac{1}{1000}$ in.; there is no apparent difference except in size; there is a well-marked intestine with cesophageal bulb, also some differentiation of the body protoplasm into reproductive organs (ovary and testicle), but I have not been able to make out any sexual difference.

The young filariæ are generally only to be found in the thorax, yet a few occur in some instances in the abdominal cavity. There are usually three or four

¹ Trans. Linn. Soc., Lond., vol. ii., part x., Zoology, p. 367.

² Proc. Linn. Soc., March 7, 1878. China Customs Medical Reports, September, 1877.

filariæ present, sometimes as many as twenty-five. In twenty filarated mosquitoes that were killed and examined between sixteen and sixty days, every one of them contained actively moving filariæ.

Mosquitoes bearing filariæ do not appear to be injured seriously; one that was killed fifty days after its meal of blood contained eleven filariæ in the thorax and two in the abdominal cavity.

In mosquitoes fed on non-filarated blood no filariæ could be detected.

When the mosquito's thorax is torn across several times with dissecting needles in a watch-glass containing water on the stage of a dissecting microscope, the filariæ are liberated and sink to the bottom; they can be seen fairly easily with the naked eye and by aid of a needle picked out; they cannot swim nor move away from the spot where they happen to sink, yet they twist and wriggle about in a violent manner; by means of what appear to be caudal suckers some of them stick to the glass, also to the dissecting needle when touched by the same.

Water is injurious to them, for after three or four hours therein they die. Water therefore cannot be the medium, as was generally supposed, by which they ultimately reach the human subject.

Directly after having seen the first "actively moving filariæ" wriggling about in water for a couple of hours, I concluded that water was the medium, and wrote a letter to the editor of the *Australasian Medical Gazette*¹ to that effect, being anxious to correct a former statement² of mine to the effect that the young filariæ died in water (as subsequent observation has shown that statement did not require correction). Shortly after having written the letter I found the young filariæ were dead, but concluded that they must have been injured by the cyanide of potassium by which the mosquito was killed. Many experiments were afterwards made with filariæ from mosquitoes that had been dissected whilst alive to insure no injury to the filariæ they contained; it made no difference, however, for the young filariæ always died after three or four hours' immersion in water. In mosquitoes that had died a natural death, when examined twenty-four hours afterwards, the filariæ were dead; this occurred whether the mosquito died on water or not. The filariæ never escape naturally from the mosquito's body.

In order, therefore, for the young filariæ to reach the human subject, it would appear that the mosquito must be swallowed. It is not uncommon to meet people who have accidentally swallowed one of these insects, and it seems possible enough that such might occur, especially in those who sleep with the mouth open. In the act of killing mosquitoes with the hand their bodies are ruptured, and any young filariæ that might be present would be extruded on to the fingers and afterwards transferred to the mouth. Mosquitoes when aged frequently get bogged in jam and honey, and by such food it is possible, although somewhat improbable, they could gain entrance into the human stomach. To be infected some may imagine that there must be an easier way than by swallowing

mosquitoes. They must remember, however, that Nature has not ordained that the life-cycle of entozoal parasites shall be easily attained; obviously for the reason that were it easily accomplished, gross infection would occur causing the death of the host and with him the parasites.

Leuckart in his work³ makes the following reference to Manson's discovery, p. 64, footnote:—"From the observations of Manson² there can no longer be any doubt that the few embryos which can pass without danger to themselves through the intestine of the mosquito undergo further development in the body-cavity, in consequence of which they now differ in size and in the structure of the mouth parts from the embryo at an earlier stage. Manson is of opinion that embryos, having thus reached a certain stage in the body-cavity, get into water only on the death of the host, and that they are taken into the human body with the water. This statement still requires demonstration, but even were this proof forthcoming there would yet remain a possibility that the embryos evacuated with the urine (which probably no more represent useless production than the eggs of intestinal worms which pass out with the fæces) may be transported to certain small hosts, and by these means human beings may perhaps be infected more commonly than in the way pointed out by Manson."

From these remarks it would appear that Leuckart imagined that it was a normal occurrence for embryo filariæ to pass out of the body with the urine; such is not so, however, and is by no means common in those affected with filariasis; it occurs in cases only when there is rupture of a lymphatic or blood-vessel in the kidney or bladder; the filariæ when mixed with urine are rapidly altered by endosmosis or exosmosis and live but a short time. The same applies to dogs affected by the *Filaria immitis*, in which, however, it is even of rarer occurrence.

How did it come about that Manson saw the final stage of the metamorphosis in mosquitoes seven days old?

This I believe to be the explanation:—The filarated mosquitoes upon which he made his observations were not bred out, and thus in confinement from the moment of their emergence from the pupa state; they were free mosquitoes obtained from a room where filarated persons slept. A few of the mosquitoes that were captured doubtless had imbibed blood weeks before, and already contained advanced stages of the metamorphosis. They were imprisoned and never fed, consequently they died about the sixth or seventh day, when they were microscopically examined. Manson evidently believed that their last meal of blood was their first.

Manson has remarked⁴ "that various stages of the metamorphosis were occasionally to be seen in the same mosquito." Such a thing never occurred to me, and is inexplicable except on the supposition that his mosquitoes had imbibed filarated blood on several different occasions.

¹ *Australasian Medical Gazette*, March 20, 1899.

² *Ibid.*, June 20, 1898.

³ "Parasites of Man," by Rudolph Leuckart. Young J. Pentland, 1886.

⁴ *Trans. Linn. Soc., Lond.*, pp. 367-8, 1884.

⁵ *Op. cit.*, p. 379.

In the following details my observations differ from those of others who have worked at this subject:—

(1) Pressure of the cover-glass more particularly and endosmosis are the cause of rupture and escape of material at the anus in the young filariæ; such is not a natural phenomenon; it will not happen if the thorax be teased out in Müller's fluid and examined without a cover-glass or with a small piece of cover-glass.

(2) After the meal of blood is digested, the mosquito's stomach and intestine contain no filariæ.

(3) The filariæ, after imbedding themselves in the thoracic muscles, lie quiescent until about the fourteenth or fifteenth day, when very slight movements can sometimes be detected.

(4) I have been unable to satisfy myself that the embryo filariæ cast their sheaths before leaving the mosquito's stomach; when seen in the thorax they appear to have lost the long collapsed sheath following tail. The sheath may, however, have only shrunk, or it may be filled out by the worm, which has already grown longer and thicker. The tail is peculiar in the early stages, which may be due possibly to retention of the sheath.

(5) The filariæ, which emigrate to the thorax, do so directly they are withdrawn from the human host; those that are to be seen in the mosquito's stomach several hours later are they, that for some reason, whether being too young, or from injury or from having been already acted upon by the digestive juices, are not destined to enter upon a metamorphosis. Loss of sheath, striation of body, changes in the body protoplasm in them are due to endosmosis and digestion.

(6) No apparent sheath can be seen in the embryo filariæ in freshly drawn blood, but a flagellum-like body generally following the tail. Sometimes the flagellum-like body is momentarily protruded from the head, and *pari passu* disappears from the tail; this only occurs when the worm is swimming tail first. Such appearance cannot be seen in every embryo, and I am inclined to think that it is not normal. The flagellum-like body is the collapsed sheath, which can only be diagnosed as a sheath when endosmosis has taken place. Those who have figured the embryo have represented a worm inside a distended sack; such appearance is unnatural. The purpose of the sheath is possibly to anchor the worm to the side of a blood-vessel when the latter is resting.

Manson, in his recent work¹ (p. 460), has remarked:—"It is also manifest that the purpose of the 'sheath' with which it is provided, while circulating in the human host, is to muzzle the embryo filaria, and prevent its breaking through the blood vessels, and so missing its chance of gaining access to the mosquito."

If any should care to decide the question for himself, let him prepare a slide of filariated blood and paint a little oil round the edge of the cover-glass to prevent evaporation, and examine under the microscope twenty-four hours afterwards, when a certain amount of coagulation and crystallisation has taken place; this forms some resistance to the filariæ, and

they may be seen crossing from one edge of the cover-glass to the other in a tortuous but definite course, with the collapsed sheath following tail.

I cannot agree with Manson that the sheath muzzles or impedes the filaria in any way; normally, I believe the sheath is never separate from the body. The embryo in freshly drawn blood wriggles about, but never seems to leave the same spot. This peculiarity was considered due to some impediment caused by the sheath, but the embryo of *Filaria immitis*, which is not possessed of a sheath, wriggles precisely in the same manner.

(7) Some writers would lead you to imagine that there is but a single pair of adult filariæ in each filariated subject. Judging from analogy of what occurs in other animals harbouring filariæ, I believe that there are generally a good many present, a dozen or so, or possibly in some cases fifty. The number of embryos that are to be found in a drop of blood is some criterion of the number of adults in the subject; if the embryos are scarce, it is likely that there are few adult females, but if plentiful it is probable there are many females.

It is not known how long the embryo filaria lives in the blood; probably it is several months, and probably the adult worms live several years.

Provided a filariated subject could prevent reinfecting himself, it is very likely that in course of five years he would be entirely free from the parasites. To accomplish this, it might be wise to emigrate to a country where there are no mosquitoes, and failing that, to sleep under perfect mosquito-net bed curtains.

Fortunately, it is easy to rid the house of the *Culex ciliaris*. It appears that this insect was introduced into Australia;¹ it will not go wild, but always frequents habitations, breeding in receptacles holding water in or about the house. Such receptacles should be covered with gauze net, perforated zinc, or other material, to exclude mosquitoes; cattle and poultry water-troughs should be emptied out at least every ten days, as by so doing the mosquito larvæ could never mature; it takes fourteen to twenty days from the mosquito egg to the perfect insect.

In this investigation the following methods were found the best. In breeding "house mosquitoes," it is necessary to obtain their eggs or larvæ. Galvanised iron washing-tubs are convenient vessels wherein to rear the larvæ; these are filled with fresh water, and placed in a shady spot; into them is put a handful of rotting leaves and a small piece of flesh, preferably flesh that has passed the putrefactive state in water, having been converted partly into adipocere. When animal matter forms part of the diet, the larvæ grow faster and to a larger size. The larvæ soon die should the water become foul. In a fortnight or so the larvæ will have changed into pupæ; by means of a miniature scoop-net (the size of a tablespoon) made of wire and mosquito net, the pupæ are transferred to a glass vessel of water, such as a fish bowl (about six inches in diameter at the mouth). The mouth of bowl is covered with muslin, the material known as "white leno" was found very serviceable; mosquito net is not suitable, as mosquitoes can, when they try, creep

¹ "Tropical Diseases," Cassell & Co., Ltd. 1898.

¹ *Proc. Linn. Soc., N. S. Wales*, vol. iii, (Series 2nd), p. 1,718.

through the meshes, especially when the net is stretched tightly. The pupæ do not require food, and in a day or two the perfect insects will have emerged from them. The male mosquitoes are easily distinguished by their large feathery antennæ; they do not suck blood. Transference of mosquitoes to a glass cell is performed by means of a "collecting tube"; this is a hollow glass cylinder conveniently four inches long and one-and-a-half inches in diameter; one end is covered with mosquito net, whilst a cork is loosely fitted to the other; pieces of Argand gas-lamp chimney make good collecting tubes.

Glass cells, about ten inches high and six inches in diameter, are convenient wherein to store living mosquitoes; they are fitted up as follows:—At the bottom is placed a little dry sand, also a vessel holding three or four ounces of water; the sand serves to weight the cell and steady the water vessel; into the vessel of water is put two or three bits of straw or cork; this is to assist the mosquitoes rising from the water; as the mosquitoes age they get infirm, and frequently get drowned, unless they reach some floating object. Over the mouth of the cell is stretched a piece of wet lino, and tied tightly with twine; when the lino is dry, a circular hole, an inch in diameter, is cut out of the centre, and this hole is covered with a watch-glass, concave side upwards.

The transference of mosquitoes to a glass cell is done in the following way:—The mosquitoes are allowed to escape under the mosquito-net curtains; the cork being removed, the mouth of a "collecting tube" is placed over a mosquito, which then flies up the tube; the cork being now replaced, the tube is brought close to the glass cell, the cork being directly over the watch-glass; the cork is removed, and the tube put right on to the watch-glass, and at the same time the watch-glass is slid aside, the open mouths of the tube and cell are now together; a puff of air blown down the tube causes the mosquito to fly down into the cell; the watch-glass is again placed in position. By such means a dozen mosquitoes might be put into a cell in a minute without any danger of injuring them.

Female mosquitoes bred out by me were put into an empty cell of the capacity of forty ounces of water, and sent to the home of the filariated subject, who liberated them under the bed curtains upon retiring; next morning, any with distended abdomen she captured by means of a collecting tube, transferred back to the cell, and returned the same to me; they were again liberated under curtains and transferred to larger vessels. In the cell storing mosquitoes a section of ripe banana is suspended; it was found best to cut the banana at right angles to its length in pieces one-and-a-half inches in length, with the skin left on. Moulds very soon grow on the cut ends, when the mosquitoes prefer to pierce the rind, and thus get at the sound tissue. It is advisable to remove the piece of banana and replace by fresh every three or four days. Should the air in the cell become foul from the decomposition of banana, or from the odour of mould fungi, or the water at times contaminated by banana juice, it is advisable to liberate the mosquitoes under a mosquito-net curtain, and transfer them to a clean cell. It is also well to place

a plug of cotton wool in the hole in lino, and over this the watch-glass, concave side down. The cells are placed in a room in the house where the light is subdued, or shaded by brown paper from too strong a light. Half-a-dozen mosquitoes is a sufficient number to put into one glass cell of the capacity of one hundred ounces of water.

When mosquitoes are required for examination, they are liberated under the curtains, captured and killed in the entomologist's cyanide bottle, or by means of chloroform, &c. Two pairs of ciliary forceps are useful with which to pull off the wings, legs, and head; afterwards the body is divided by dissecting needles into thorax and abdomen, and each portion examined separately, teased out in water, or better in Müller's fluid, with or without a cover-glass under a magnification of fifty diameters.

The following is a short account of the life-cycle of *Filaria bancrofti*:—Commencing with the mature parasites in the human subject. These are three or four inches in length by $\frac{3}{16}$ in. in breadth; they live in the lymphatic vessels; they produce the embryo filariæ, which are $\frac{3}{16}$ in. by $\frac{1}{1600}$ in. These latter live in the blood vessels, swimming about when the host is sleeping and resting themselves when he is awake.

Mosquitoes when biting a filariated subject during the night withdraw together with blood some of the embryo filariæ. Soon after the embryos reach the mosquito's stomach they pierce the stomach wall and find their way to some muscular mass, particularly the thoracic muscles, in which they imbed themselves. There nourished by the mosquito's plasma they grow at a prodigious rate, becoming longer and thicker, and assume by the fifth day an appearance in which a distinct line, the rudimentary intestine, can be seen from the mouth to the anus; the body protoplasm, at first homogeneous, has been changed into large cells with numerous vacuoles; in ten days the intestine presents a double line, the large cells have given place to very small cells. From this time on to the seventeenth day most remarkable changes occur too intricate and difficult to describe. In seventeen days thereabout the young filaria has attained its maximum development as far as its life in the mosquito is concerned. It now awaits the chance of gaining entrance to the human host; in the event of which, we presume that it will start upon a second metamorphosis, the final alternation of generations, in which it grows to the length of three or four inches and becomes sexually mature.

It remains to be proved that these young filariæ will become sexually mature in the human host. I have elsewhere¹ suggested how this might be accomplished, viz., by inducing a life-sentenced prisoner to swallow some mosquitoes bearing filariæ, on condition that he be given a free pardon.

Besides proving that the *Culex ciliaris*, Linn., is an efficient host for *Filaria nocturna*, I have shown that two other species of mosquito are not hospitable, viz., *Culex notoscriptus*, Skuse, and *C. annulirostris*, Skuse. Both these mosquitoes will live in confinement at least twenty days. *Culex notoscriptus* sucks out plenty of embryos, but, as far as I have seen, none of these ever

¹ Australasian Medical Gazette, March 20, 1899.

migrate to the thorax; they appear to have been killed by the salivary juice. Only rarely do some embryos migrate in the case of *Culex annulirostris*; after two days, however, any that did reach the thorax have died and been absorbed. Other mosquitoes have been experimented upon, but, as I have been unable to keep these alive sufficiently long for the final stage of the metamorphosis, it is impossible to say definitely that they are not hospitable, yet every thing tends to that conclusion.

In the case of *Culex hispidosus*, Skuse, and *C. vigilax*, Skuse, these two species live about seven days in confinement, and a number examined about that time contained no filariæ. In the case of *Culex nigritorax*, Macquart., *C. procan*, Skuse, and *Anopheles musivus*, Skuse, I have been unable to keep them alive more than three days. A good many experiments were made with *Anopheles musivus*. This mosquito sucks out a very large number of embryos, and the most of these migrate to the thorax.

For the scientific names of the mosquitoes I am indebted to Henry Tryon, Esq., Entomologist to the Queensland Government. Thanks are due also to E.S., the filariated subject, without whose assistance this investigation could not have been carried out, and Manson's important discovery might for some time to come have remained unbelievable.

Deception Bay,
Queensland, May, 1899.

Added June 1 :—

A number of mosquitoes imbibed filariated blood on April 26, and the final stage of the metamorphosis did not occur in them until May 31, i.e., thirty-five days. The weather was cold.

It has occurred to me that the young filariæ may gain entrance to the human host whilst mosquitoes bearing them are in the act of biting. The entrance of warm blood into the mosquito may excite the young filariæ, in consequence of which they pierce the œsophagus and pass down the proboscis into the human skin. In this way injury from the human digestive agents would be avoided. It is not unreasonable to suppose that, like water, the digestive fluids would soon kill the young filariæ, but it is probable that those that may have been set free by rupture of the mosquito's body would immediately pierce the mucous membrane and enter a lymphatic or other vessel.

PLAGUE AT SANTOS.—The *Medical Record* observes that it is stated in the *Brazil Medico* that the plague reached Santos about the middle of July, 1899, the first signs of its presence being an extraordinary mortality among the rats in one of the storehouses on the docks. Another epidemic among the rats was noted in the latter part of September, and the first case of the disease in man was discovered soon afterward in a house in the immediate vicinity of the storehouses. On October 18, the existence of the plague in Santos was officially announced.

Therapeutic Notes.

THE INUNCTION OF SALICYLIC ACID.—The *Medical News* for December 16 says that salicylic acid, if mixed with an oily vehicle, is quickly absorbed. It may be prescribed thus in connection with its internal administration for rheumatism. Oil of wintergreen may be used in place of it with advantage.

R. Salicylic acid ... 2½ drachms;
Alcohol ... 1½ ounce;
Castor oil ... 3 ounces.

M. S.: Rub into the affected part, covering first with an impervious material, then with flannel or cotton wool.—*New York Medical Journal*.

FOR INFANTILE CHOLERA.—*Presse médicale* for August 16 attributes the following to Hayem and Lesage:

R. Lactic acid ... 45 grains;
Syrup of quince ... 375 "
Distilled water ... 1,500 "

M. The solution should be iced. A coffeespoonful to be given every half hour for three hours, then every hour. Its use must be stopped as soon as the use of milk is resumed.—*New York Medical Journal*.

A QUININE MIXTURE, palatable for children, is recommended by W. J. Greanellé, who gives the following details for the preparation, which is not only readily taken by children, but also serviceable as a reward to be given after unpleasant medicines. The mixture is designed as a tonic and malarial prophylactic for children living in malarious sections. It will serve for active medication in acute cases of malarial disease in children of three years or younger, by giving the larger dose at hourly intervals. The author states that children actually crave it, and that it has frequently served as an excellent bribe, to be given immediately after a simple solution of quinine hydrochlorate in water when large doses of the drug are necessary. A teaspoonful of water carrying 2 or 3 grains of quinine hydrochlorate will be readily taken by any child, with "a spoonful of pink medicine right after." Those who have little patients in large numbers will appreciate this.

1. Quinine Hydrochlorate ... 5 to 10 grs.
Alcohol ... 1 fl. dr.
2. Oil Cinnamon ... } of each, 30 to 40 min.
Oil Anise ... }
Magnesia ... sufficient
Water ... 1 fl. oz.

Let stand for some hours; filter.

Mix 1 and 2 and add—

3. Simple syrup ... 3 fl. oz.
Carmin or Cochineal Solution ... 5 drops.

Dose: 1 or 2 fl. dr. as directed.

Saccharin in small quantity helps to disguise the larger dose of quinine. Small doses of Fowler's solution may be added, if indicated, or sodium bromide for children made irritable by quinine.

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THE

Journal of Tropical Medicine

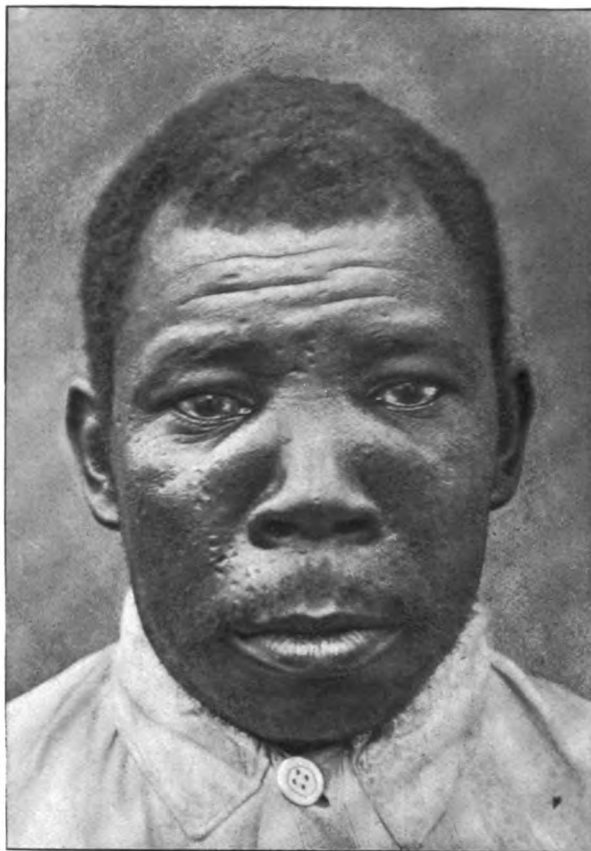
JANUARY, 1900.

THE WAR AND DISEASE.

THE stress of war is already beginning to make itself felt in the British Army in South Africa. Enteric fever and dysentery are appearing in the reports, and especially so in those of the beleagured camp at Ladysmith. It is hardly three months since the war begun, and it is only midsummer in the region of the war operations; so that the early appearance of these diseases is ominous of what is likely to occur in the autumn. That affairs in this respect should be worse among the troops under General White is only what might be expected, for, notwithstanding the fact that there is no lack of provisions, and that the troops are in good spirits, the close aggregation of men, on a necessarily limited space, under

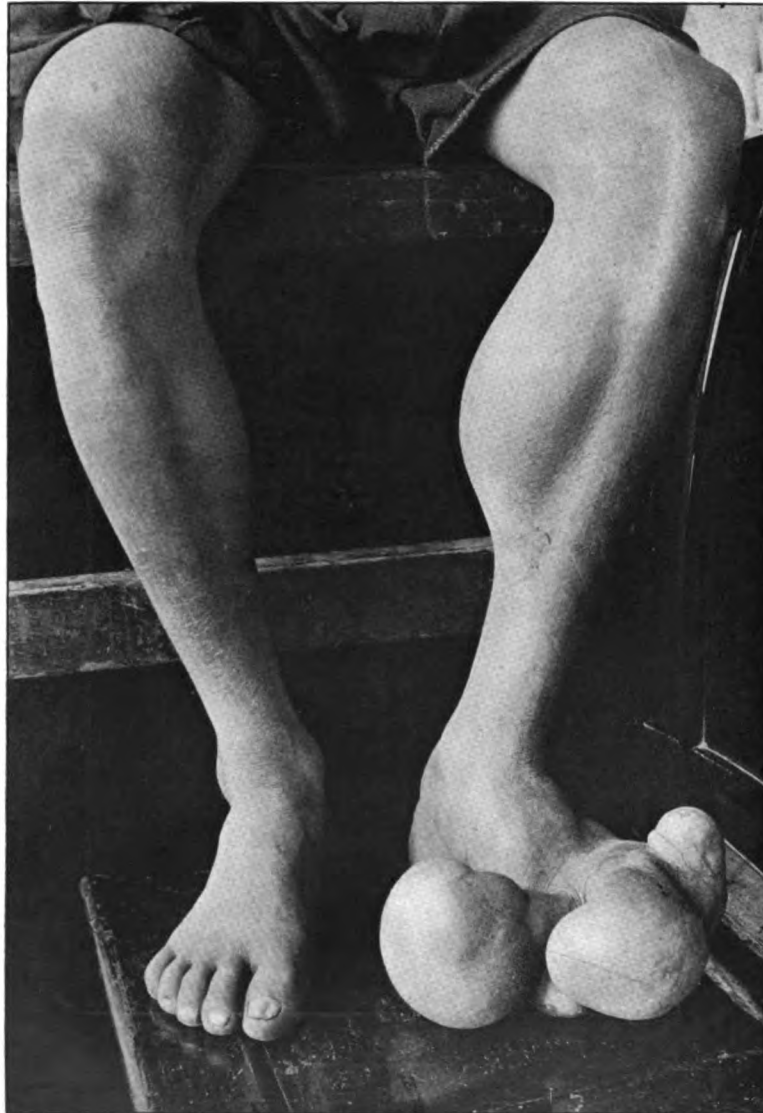
the conditions of a siege, must sooner or later end in the development of diseases of camp-life. The lessened amount of sleep, the difficulty of obtaining pure water, combined with the primitive arrangements for conservancy; the pollution of soil by men, horses, and cattle; the burial of the dead not far from the living; and the disposal of dead carcasses of animals, are conditions making for disease, and only time and season are required to make a camp unhealthy. How far the best means have been adopted to ward off as far as possible the evils likely to arise from these circumstances, and to limit the prevalence of disease, is not known. We hope they have been fully appreciated beforehand, and that an undue optimism has not allowed the mistake to be made of underestimating the consequences of any slackness in these matters; but that they have been well considered, and that every precaution which science dictates, and which the unusual circumstances will permit, has been taken.

This is a war which, as Lord Rosebery has said, is no small war. The experience of the past three months lead us to believe that it is likely to be a long war, and accordingly different in many respects from the short and comparatively healthy campaigns with which the present generation is familiar. It will test the resources of sanitary officers, whose duties will be as laborious as they are important. It was with much satisfaction we heard of the appointment of Captain Hughes, R.A.M.C., to General Buller's staff, and it is with profound sorrow we hear of his death on the battle field. His loss is more than a personal one to his friends, because it is particularly unfortunate to the army in Natal, which is deprived of an officer whose sound sanitary knowledge and experience would have been invaluable. There are many other respects in which the conditions of South Africa are full of anxiety at the present moment. The season of the year is not as propitious for health as we should like it to be. We read of drought in the East and scarcity of crops in the Orange Free State, both of which require careful attention and vigilance on the part of sanitary officers, for with the disorganisation of



A CASE OF GOUNDOU OR ANAKHRE.

From a Photograph illustrating Dr. W. RENNER's article in the current issue.



PES GIGAS.

From a Photograph by Dr. P. B. COUSLAND, Swatow, China.

Patient was a young Chinaman and the disease was congenital. The affected parts felt like normal tissue.
The condition of the calf of the diseased limb is seen to be greatly hypertrophied.

those parts of the country which are filled with troops, with war and with scarcity, there is every probability, if the utmost care is not taken, of pestilence adding its horrors to the situation. It is necessary to look this possibility in the face, not for the purposes of sensational alarm but in order to be prepared for all contingencies, and in order that the measures necessary shall be taken to avoid, as far as possible, such a calamity. It must never be forgotten that in the neighbourhood of Delagoa Bay there are dormant centres of plague only awaiting time, season and circumstance to become active.

ETIOLOGY OF YELLOW FEVER.

THE following important conclusions have been arrived at by Dr. Wasdin and Dr. Geddings, who were appointed by the supervising Surgeon-General of the Marine Hospital Service, Washington, to investigate yellow fever :—

(1) That the micro-organism discovered by Professor Guiseppe Sanarelli, of the University of Bologna, Italy, and by him named "Bacillus icteroides," is the cause of yellow fever.

(2) That yellow fever is naturally infectious to certain animals, the degree varying with the species; that in some rodents local infection is very quickly followed by blood infection, and that while in dogs and rabbits there is no evidence of the subsequent invasion of the blood, monkeys react to the infection the same as man.

(3) That infection takes place by way of the respiratory tract, the primary colonisation in this tract giving rise to the earlier manifestations of the disease.

(4) That in many cases of the disease, probably a majority, the primary infection or colonisation in the lungs is followed by a "secondary infection," or a secondary colonisation of this organism, in the blood of the patient. This secondary infection may be complicated by the co-instantaneous passage of other organisms into the blood, or this complication may arise during the last hours of life.

(5) There is no evidence to support the theory

advanced by Professor Sanarelli that this disease is primarily septicæmia, inasmuch as cases do occur in which the *Bacillus icteroides* cannot be found in the blood or organs in which it might be deposited therefrom.

(6) That there exists no causal relationship between the bacillus x of Sternberg and this highly infectious disease, and that this bacillus x is frequently found in the intestinal contents of normal animals and of man, as well as in the urine and the bronchial secretion.

(7) That, so far as the Commission is aware, the *Bacillus icteroides* has never been found in any body other than of one infected with yellow fever; and that whatever may be the cultural similarities between this and other micro-organisms, it is characterised by a specificity which is distinctive.

(8) That the *Bacillus icteroides* is very susceptible to the influences injurious to bacterial life, and that its ready control by the processes of disinfection, chemical and mechanical, is assured.

(9) That the *Bacillus icteroides* produces *in vitro*, as well as *in vita*, a toxin of the most marked potency; and that from our present knowledge there exists a reasonable possibility of the ultimate production of an anti-serum more potent than that prepared by Professor Sanarelli.

It is with much pleasure we see among the list of New Year's honours a larger number than usual of medical men especially connected with the Colonies. There is, first of all, Dr. Manson, whom the Queen delights to honour with the order of St. Michael and St. George. It is in recognition of his splendid work, which has gained for him a world-wide reputation. As medical adviser to the Colonial Office, this decoration is the first step, we have no doubt, to a still higher honour. The year has begun well with him and with the other medical men who have had honours bestowed on them; and we wish them, one and all, "A Happy New Year," and many years of health and strength to enjoy the confidence of the Queen.

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

WITH reference to your "Article for Discussion" in the JOURNAL for October, viz., "The Rarity or Absence of Rheumatic Fever in the Tropics," I should like to make the following observations on the subject.

During my eight years of hospital practice in the Straits Settlements (Penang and Singapore) and in the Malay Peninsula, I have not come across a single case of rheumatic fever in a native and only one case in a European. This European had suffered from the disease in Britain, and had had previous attacks in the Straits.

He was in Penang at the time I attended him for rheumatic fever, and during the two years I was there he only had this one attack. He had no cardiac lesion, and the attack was a very mild one.

Clinically, I have observed, though very rarely, valvular cardiac lesion in natives (Chinese and Indians, not Malays), but could never elicit a previous history of rheumatic fever.

In the *post-mortem* room, on examining bodies of natives brought by the police, I have occasionally found chronic valvular lesions of undoubtedly rheumatic origin.

In my experience, the more chronic forms of rheumatism, as seen in Britain, are rare in the Straits Settlements and Malay Peninsula.

I should also like to mention here that I have never met with a case of scarlet fever in the Straits or in the Malay Peninsula.

A. J. McCLOSKEY, M.B.,
District Surgeon.

Erlangen, Malay Peninsula.

I AM glad to be able to add something to the subject of "Acute Rheumatism in the Tropics," introduced for discussion in the JOURNAL for October. I have a young tea-planter, a four years' resident in the country, who has had three attacks of rheumatic fever since his arrival.

Some time before leaving home he passed through what appears to have been a very severe attack of acute rheumatism. He was not medically examined before coming out, else he would have been rejected, for I suspect even at that time his heart was affected.

During the first cold weather after his arrival he had a recurrence of the fever—suffered from painful joints, swelling of feet and legs, had cardiac pains and the like; at this time he was not under my care. Next cold weather the same thing happened; I saw him early, found that he was threatened with rheumatic fever, gave him salicylate of soda, and he was soon well. Last year, towards the end of the cold weather, he again began to suffer from sleeplessness, horrid dreams, pains over heart and other symptoms indicating a recurrence of his fever. Rest, salicylates, belladonna, change of air and tonics saved him from an actual attack, but he was weak for a considerable time. His heart has not escaped during these attacks; there is narrowing at both the mitral and aortic orifices, but compensation is complete, and he can manage his work satisfactorily.

These occurrences had no connection with ordinary malarial fever, from which he also suffers from time to time; but along with his quinine he very often takes a few grains of salicylate of soda as a safeguard.

Rheumatic fever amongst the natives is extremely rare in my experience; most of what at first sight appears to be rheumatism turns out to be gonorrhœal synovitis.

A. B. DALGETTY, M.B., C.M.

South Sylhet, India,

November 20, 1899.

THE QUEEN has been pleased to confer the following honours on the occasion of the New Year:—Thomas Lauder Brunton, M.D., F.R.S.; Surgeon-General F. R. Lovell, C.M.G., M.E.L.C., Trinidad, have had the honour of knighthood conferred on them.

THE Honourable John Alexander Cockburn, M.D., formerly Premier of South Australia, now Agent-General in London for that Colony, has been made K.C.M.G.

Translations.

FEBRIS BILIOSA HÆMOGLOBINURICA
(BLACKWATER FEVER) AND QUININE
INTOXICATIONS IN NETHERLAND-
INDIES.

By Dr. I. H. F. KOHLBRUGGE.

Translated by P. FALCKE.

ALTHOUGH it is a long time ago that Barthélemy-Benoit described this malarial fever, it has lately aroused more general interest, because in the newly acquired colonies, particularly the German ones and that on the Congo, it has shown itself to be especially virulent, and has thus given occasion for many opinions through the medium of German technical publications.

First of all, I must call attention to the fact that, notwithstanding large malarial practice (2,000 patients), I have never seen a case of blackwater fever, and the reason I permit myself the liberty of writing a few lines on this disease is that I thought it worth while to go through the literature of the subject as to the occurrence of the disease in the Malay Archipelago, and the result gives me the opportunity to communicate my experience as regards quinine intoxications and the prophylaxis of the same.

Febris biliosa hæmoglobinurica first came to general knowledge by means of the first Atjeh war (1874 to 1878), during which, also, the well-known beriberi epidemics were originated. At Atjeh, however, the disease occurred almost solely in two small forts, Tjade and Toengkoep. From this fact alone it may be gathered that it is a disease *sui generis*, and cannot be said to be due to quinine intoxication. There were earlier observers (Jacobs) who were also inclined to dissociate it from the other malarial illnesses. The notorious island Anrust, and the equally notorious harbour Tjilatgap were other seats of this fever, but isolated cases occurred at Magelang, William's Isle, Semarang, Soerabaya, and Macassar.

We are indebted to Dr. J. Jacobs for an exhaustive treatise on this fever. After having observed twenty cases he came to the conclusion that the symptoms much resembled those which Barthélemy-Benoit has described. The illness always set in very acutely and ran its course quickly; the fever was always remittent, never intermittent; furthermore, he observed severe prostration, hæmoglobinuria (without inflammation of the kidneys and bladder), slight albuminuria, which was connected with the hæmoglobinuria, a total lack of gall-pigment in the urine, slight icterus, which in a few patients was limited to the conjunctivæ. At the autopsies the following observations were made: a very soft, swollen spleen, great impoverishment of the blood, blood without any coagula of fibrine; the liver was always normal, the ductus choledochus free, the gall-bladder filled with very thick, but fluid, dark brown gall. Most of the patients died. Furthermore, the work contains an exhaustive discussion on the questions as to whether febris biliosa is to be classed with the malaria fevers, and whether the icterus be of a hæmotagenous or hepatogenous character. As to the latter question, we shall return to it later on.

The acute appearance of the fever in patients who had previously been healthy, with the immediate setting in of hæmoglobinuria, shows that in the greater number of the cases quinine intoxication may certainly be excluded, and in the few others is open to doubt.

In most patients the fever decreased slowly. Five recovered. Of these one had had two attacks (intervals not given), and another had had three attacks, with an interval of seven months between the first and second, and five weeks between the second and last. Each time the attacks were favourably influenced by immediate change of locality.

It has been mentioned above that Jacobs never observed enlargement of the liver. In one patient, whose case had a fatal termination, hypertrophy of the liver was only discovered after death, but, as connective tissue cords were found on the same, the hypertrophy may have existed long before the last illness (Grondhout).

In another case (Waschke) the fever and hepatic enlargement were simultaneous and acute, and there was much pain in the hepatic region. As a rule, however, the liver is normal.

With one exception (v. d. Scheer) the spleen was always enlarged, and some patients had severe pain in consequence. In some cases the icterus was very pronounced, in others slighter. Most authors assert that the icterus tallies with the fever, rising and falling with the same; the increase was regular during the first days.

The observation on the fæces of eight persons yielded very important results. Four patients exhibited frequent evacuations; of three of these the good colour was mentioned, and in the fourth a bilious hue. The other four showed bloody stools, so that they also lost blood this way. The intestinal hæmorrhages mostly lasted from three to six days. Several of my old colleagues inform me that intestinal hæmorrhage is often associated with hæmatemesis in febris biliosa hæmoglobinurica.

Hæmoglobinuria was mostly very pronounced; the colour of the urine was dark red. As with the icterus, the hæmoglobinuria was connected with the fever, increasing and decreasing with the same. After a few days the urine, in four patients, became brownish-red (beer-colour), or light brown, and then the abnormal colouring disappeared. The urine never contained gall-pigment; blood corpuscles were never found in the urine; there was always a reaction of hæmoglobin, and mostly reaction of albumen.

It is mentioned that four patients suffered with nausea and vomiting; three of them also complained of a bitter taste in the mouth, and vomited greenish-yellow slime. An autopsy upon one of these cases showed that the gall-bladder was full, the ductus choledochus was filled with gall, but was passable. This and the normal colour of the stools proved that retention of the gall cannot be the cause of the icterus.

Blood examinations were only made in two cases. Much pigment and many leucocytes were found. Of the earlier observers, previous to 1889, van der Scheer alone examined for plasmodia, and found them plentiful.

Most patients complained of great weakness, and

suffered with anæmia. Van der Scheer's patient alone was drowsy, delirious, and exhibited incontinence of bladder and rectum.

All the patients, even those of Jacobs, were treated with large doses of quinine, and not one author mentions that the drug had a deleterious effect; many recovered completely notwithstanding an uninterrupted course of quinine. Only one author mentions that the quinine was vomited (this often occurs in malaria, also with calomel), and he therefore substituted salicylate of soda (three cases).

On the ground that the liver was mostly not enlarged, that engorgement of the gall bladder was always lacking, and that gall pigment was never observed in the urine, Jacobs founds his opinion that the icterus must be of a pure hæmatogenous nature, and cannot be hepatogenous. He also differentiates the febris intermittens perniciose icterica from the febris biliosa hæmoglobinurica.

The bloody stools (and the hæmatemesis) point to a universal affection of the vascular system, which is not unknown in malaria as well as in blackwater fever. In malaria hæmorrhages are often seen; those best known are the intestinal hæmorrhages (from the colon or rectum) which I myself have often observed. In the stools, besides fæces one finds a great mass of blood without admixture of slime or shreds of tissue. Furthermore hæmoptysis is known (without inflammation of the lungs), also bleeding of the nose, menorrhagia and petechia in the skin. Such a case of purpura through malaria has been described by Waschke, and could not be ascribed to quinine intoxication, as the spots appeared before any drugs at all had been given.

If one takes note that malaria has the effect not alone of dissolving blood, but of increasing the porosity of the capillaries, one can scarcely regard blackwater fever as a special form of malaria. Why malaria attacks at one time one organ and at another time a different one is beyond our knowledge, but the same holds good in regard to all other illnesses influencing the whole body. Previous weakness, personal predisposition, particularly violent infection, must be called to our aid to explain this; but other causes, also, must co-operate, such as a variability of the plasmodia in certain localities, whereby the appearance of hæmoglobinuria on sharply circumscribed spots takes place (the garrison of a fort). One must then consider the likelihood of a simultaneous infection, or call to one's mind meteorological factors, and in this direction it is worthy of attention that blackwater fever is always seen in those places that are known to be particularly unhealthy.

In regard to therapeutics and the deleterious effects of quinine the following is worthy of attention.

At the time that blackwater fever was frequently observed in the Malay archipelago, quinine was never given in such large doses as are prescribed at the present day. Many doctors also avoided quinine in remittent fevers. If quinine originated hæmoglobinuria, blackwater fever would be now observed much more frequently than formerly, whereas it has become very rare in this archipelago.¹ Quinine can

therefore not always be the cause of hæmoglobinuria. It cannot, however, be disputed that hæmoglobinuria, when already extant, can be aggravated by quinine; nor that in persons with a certain idiosyncrasy it may originate hæmoglobinuria. I myself have seen œdema, erythema and petechia, after use of quinine, as also metrorrhagia. Quinine in these cases acts on the capillaries.

We thus are on the horns of a dilemma. Malaria and quinine may originate hæmoglobinuria, which is so weakening to the patient, but we cannot possibly do without quinine, especially as febris biliosa hæmoglobinurica has undoubtedly often been cured by quinine, just in the same way as I myself have cured with the same drugs the hæmorrhages occurring in malaria.

Though hæmoglobinuria is very seldom caused by quinine, still we must admit that in a few persons quinine does have that effect. Every tropical practitioner has come across patients who could not take quinine. Should such patients in addition be cachectic, then quinine is to be contra-indicated, once and for always, and the patient treated by change of locality. Should the fever be of a remittent character, then it is also better to eschew quinine.

With an extensive malaria practice the tropical practitioner will, however, find by experience that, even with those patients with pronounced idiosyncrasy and in spite of cachexia, quinine cannot be done away with, as, for instance, with a pure intermittent or in a case where change of locality is out of the question. What is he then to do?

I have some years ago solved this riddle, for I found that it was only those preparations of quinine prepared with inorganic acids that originated intoxications, but that the tannate of quinine could be taken by persons possessed of a marked idiosyncrasy, in the largest doses, without any evil effect, particularly if the astringent effect (not always desirable) was neutralised by suitable diet, massage, or other means. I prescribed tannate of quinine to all those patients of mine who could not take ordinary quinine, occasionally up to about fifteen grains, and always in wafers.

In support of my method I will add the history of a case. The influence of quinine on the pregnant uterus is well known; it is therefore usual only to give quinine after incomplete abortion, in order to expel any parts that remained.

I treated a lady who, at her first pregnancy, got hæmorrhage through quinine and aborted. On again becoming pregnant she again had malaria, and on account of her previous sad experience she was willing to bear the severest fever rather than take quinine. As, however, fever is likely to cause abortion, I gave her daily four to six gramm tannate of quinine; she recovered, and bore a healthy child at term.

In the same way, all those persons who suffered with œdema or petechia through taking the sulphate, hydrochlorate, or hydrobroma of quinine could bear tannate of quinine excellently well. I may, there-

¹ Of 2,000 patients suffering with fever, Burot and Legrand (Madagascar) only observed three cases of blackwater fever.

In the archipelago of the Dutch Indies, the disease has just re-appeared at Lombok Island, which is the unhealthiest garrison.

fore, advise my colleagues who, in Africa, so often have the opportunity of treating blackwater fever to in future give their patients who may be suffering with this illness tannate of quinine in large doses.

One should also in future not condemn all quinine preparations of causing intoxications; but one should distinguish between the intoxications of the various quinine salts of which the tannic-acid salt is the least pernicious.

Tosari-Java, July 23, 1898.

INTERTROPICAL PATHOLOGY. ON THE PART PLAYED BY INSECTS IN THE DISSEMINATION OF THE DISEASES OF HOT COUNTRIES.

By Dr. E. CALMETE.

A STUDY has been made for some years of the part played by insects in the dissemination of infectious disease. Putting aside the opinion of travellers and doctors, whose inconclusive observations are open to doubt, very few experiments remain which have been verified in the laboratory by authorities upon the subject. It is evident, however, that for medical men in hot countries some interest will be found in a brief analysis of the facts collected by positive science upon such subjects.

It is well known that popular tradition, nearly always the precursor of scientific discovery, recognised flies as the disseminators of carbuncle, cholera and plague; this belief has proved correct in regard to the first-named disease. Davaine, on the strength of irrefutable experiments, proved the share of certain diptera in the dissemination of carbuncle. Magnin, a capable entomologist, discovered the species which enjoys this unfortunate privilege. However, the transmission of carbuncle is not generally effected through the agency of flies. It is necessary that the economy should give the bacterial spores an opportunity of penetrating into the organism. Existing experiments tend to show that flies are capable of transmitting cholera. Simmond (of Hamburg) whilst performing an autopsy on the body of a cholera patient, noticed a large number of flies in the neighbourhood of the intestines; placing some of them for the space of an hour in a vessel, the sides of which had previously been covered with gelatine, three days afterwards he verified the development of numerous colonies of cholera bacillus. Yersin succeeded in transmitting plague to Indian pigs, which he inoculated with water containing dead flies collected in the laboratory where animals had been inoculated with the plague. Dr. Simmond proved in an interesting paper that fleas are the chief agent of infection in this terrible disease. (*Annales de l'Inst. Pasteur*, 1898, pages 625-688.) This eminent colleague, having in view different epidemical foci, noted the following facts:—

The natives, who carried by the tail a rat, which had died of the plague a few hours before, nearly always succumbed to that disease four or five days later. On the contrary, twenty-four hours after the death of the rat there was no danger in touching it.

Under these circumstances, Simmond asks:—How and why should the animal spread contagion a few hours after its death, and prove innocuous after twenty-four hours? The explanation is simple, taking into consideration the new method of spreading infectious diseases hitherto unknown. Healthy rats free themselves from fleas by eating them; but when they are sick they take no notice of them, and the fleas only abandon them when their body is quite cold. This explains why a person carrying a rat recently dead of the plague is at once attacked by the disease, the fleas passing on to his body from that of the rat; but this may be done without peril twenty-four hours after the death of the animal, the fleas having migrated in the interim.

This explanation of the spread of plague contagion by inoculation is the more acceptable as the disease often manifests itself by phlyctæna, the serum of which is a pure culture of plague bacillus. For further details we refer our readers to Dr. Simmond's paper.

We will not abandon the study of the part played by flies in the transmission of parasitical affections without referring to the experiments of David Bruce with regard to *Nagana*, an affection which attacks horses and dogs, and is caused by the fly *Tsé-Tsé*. This author also succeeded in determining the disease far from its ordinary forms, causing the horses to be stung by flies collected from the original focus. We must also remember that Spillmann and Haushalter (of Nancy) found the bacillus of tuberculosis in the abdomen of flies which had a predilection for settling on the lips of phthisical patients. Fleas and flies are not the only parasitical insects which have been recognised as the disseminators of infectious diseases. Bugs have also been suspected of carrying the infection of cancer, and especially of relapsing fever.

We will first concern ourselves with this last disease. It is known to be due to a spirillum in the blood of the patients, discovered by Obermeier in the year 1868. In 1897, Dr. Tiktine, studying a great epidemic of relapsing fever which raged persistently in Odessa, was struck by the fact that the victims were for the most part inmates of night refuges. His investigations with regard to lice as agents of contagion yielded negative results; but his attention was principally drawn to the important part played by bugs in this matter. Placing a certain number of these insects in a kind of cupping-glass, he applied them to the feet of patients suffering from recurrent fever, in whose blood the presence of spirilla had been positively ascertained. When the parasites were full of blood he killed them, and found in all the germs of relapsing fever. Tiktine also inoculated various animals with the spirilla obtained from the infected bugs, and after the lapse of sixty hours these animals not only exhibited the symptoms of typho-malarial fever, but he also found their blood invaded by pathogenic spirilla.

The experiments of the Russian doctor are very suggestive; in opposition to the assertion of Nuttal, they prove that the germs of contagion absorbed by the bugs may preserve their virulence, in spite of their contact with the digestive juices of these insects. As to the part played by bugs in the spreading of cancer,

we are inclined to accept the facts related by Morau. These are so exact that they give rise to great hopes that the result will be the discovery of the etiology of this disease.

This skilful experimenter isolated healthy rats in a completely aseptic cage, afterwards introducing bugs taken from another cage containing cancerous rats, and after a few months he observed that nearly all the rats in the cage infected with the bugs were attacked by cancer.

Does not this experimental fact, as we explained it in 1896 (*Bull. Med.*, p. 1,229), prove the possibility of the spread of cancer by foreign organisms in the interior of which the cancer germ is elaborated? Is it not by the study of coccidia, parasites of intracellular evolution, alternating generation, and undefined transformation, that such questions will at last be completely elucidated?

The recent discoveries of Dr. Ronald Ross concerning the part played by mosquitoes in the transmission of paludism, prove that these discoveries are applicable to a much wider field than is supposed. We will state in detail the facts asserted in Ross's memorandum (*Annals of the Pasteur Inst.*, 1899, p. 136).

The hæmatozoa of paludism undergoes actual transformation in the body of the mosquito, passing through successive phases necessary for its complete evolution. It is known that Laveran, from the beginning of his studies, advanced the opinion that mosquitoes played the same part in the transmission of paludism as in that of filariasis.

The learned Italians, Bignani, Dionisi and Grassi, wishing to determine the species of mosquito which serve as powerful intermediaries for the malarial parasite, pointed out for the consideration of the medical world, that the large-sized mosquitoes are frequently found in countries where malaria is prevalent, and known to zoologists as *anopheles claviger*.

Grassi deduced a frequent relation between the presence of the mosquito and outbreaks of paludal fever not only in Lombardy, but also in Venice, the morasses of Tuscany, and the Campagna of Rome.

The *anopheles* are characterised by the presence of four white markings on the wings disposed in the form of a T. In marshy places it is also easy to discover the *culex penicillaris*, *Richiarti*, *hortensis*, &c. These mosquitoes sting principally in the morning and at night, from June to September, which are the most dangerous months. They readily invade low buildings, but rarely penetrate to the upper stories of houses. In the state of larvæ and nymphæ they are found on the surface of the water, chiefly stagnant, head downwards. If disturbed by agitation of the water or by any noise, they sink rapidly into the depths, their bodies taking the form of the arc of a circle. They undergo three metamorphosis after they are transformed into nymphæ, and continue to live in the water. The water furnishes for their sustenance the residue of vegetable matter existing in suspension. On passing from the state of nymphæ to that of adult insects they become hæmatophagous, and attack man and animals. The female of the mosquito produces in a short time an innumerable quantity of eggs, the mass of which float upon the water in the

form of rosaries. In the space of from four to six weeks these eggs produce as many adult mosquitoes, ready in their turn for further reproduction. Such are the habits of these singular agents in the transmission of paludism.

Let us now consider the after-life of the hæmatozoa. Ronald Ross, causing birds to be stung by mosquitoes of a determined species, which had previously been infected with the malarial hæmatozoa, made the following observations:—After a certain time the hæmatozoa became impregnated with pigment, and were converted into a pigmented body; after this they grew, and adhered to the external involucre of the stomach of the mosquito, and there losing their pigment, passed into the circulation of the insect's blood. In this conjuncture they give birth to two kinds of reproductive elements: (1) filiform elements 16 micromillimetres in breadth; (2) black spores, showing large bodies of a dark grey colour and varying curvature.

Now what becomes of these micro-organisms? The black spores remain in the insect, where they grow and reproduce forms capable of vegetating in the interior of the said insect. The filiform elements, however, are found in great quantities in the poison-salivary glands, from whence they pass with the greatest facility through the sting of the mosquito into the skin of the healthy individual. Such, according to Ross, is the manner in which paludism is transmitted. This skilful observer succeeded in infecting by this process innumerable sparrows previously exposed to the stings of infected mosquitoes. He observed in all the birds a period of incubation of from five to eight days, after which the hæmatozoa multiplied rapidly until from ten to sixty could be counted in the microscopic field. The greater number of birds died with a characteristic pigmentation of the liver.

In man the complete cycle of evolution of the hæmatozoa is similar to what it is in birds, and is resumed in the following phases: The absorption of the hæmatozoa by the mosquito; its growth and adherence to the walls of the insect's stomach; the production of germ filaments (analogous to flagella); the penetration of these filaments into the poison-salivary glands, and finally the dissemination of these germs into the capillaries of the healthy individual by penetration of the skin.

Ronald Ross says, however, that there is still much to be done. All the harbourers of human parasites in countries where malaria exists, should be discovered, and the pathogenic value of the black spores described by the author determined, which in the absence of more certain information seem to produce germ filaments outside the organism in stagnant waters. Such, at least, is the opinion of Manson (*British Med. Journal*, 1898). In any case it is evident that our knowledge of the positive conditions of paludism has taken a great step forward since our biological and zoological studies have been taken up in France, Italy and England.

We await with confidence the result of the investigations which are actually being carried on in the African continent, the exploration of whose riches will only be made possible by the disappearance of malaria.

A. E.

Recent Literature on Tropical Medicine.

SCARLATINA IN INDIA.

By ARNOLD CADDY, F.R.C.S.Eng., *Member of the Clinical and Pathological Societies of London*; and J. NIELD COOK, D.P.H.Camb., *Medical Officer of Health, Calcutta*.

Two cases of a disease simulating scarlatina very closely having come under our notice, we have thought fit to record them in view of the freely expressed opinion that scarlatina is unknown in India, except in a few instances where it has been directly imported from Europe.

Both these cases occurred in the European Female Orphan Asylum, 79, Lower Circular Road, Calcutta. This institution was founded in 1815, and the occupants are girls who have lost either father or mother or both parents. The majority of the children are of pure European parentage, and with very few exceptions have been born and bred in India.

The Asylum is superintended by two Sisters of the Order of St. John the Baptist, Clewer. When full it contains fifty-two children. The building stands in a large compound, the grounds being very well kept. There are fifteen native male servants living and sleeping in out-buildings on the premises. Two punkha coolies come in from outside during the hot weather to work by day and two *mehtrani ayahs* who come daily to sweep; these latter bring their children with them and they in no way come in contact with the European inmates.

The buildings consist of a central block of two floors, facing east, and a detached wing, facing south, situated at the north-east angle of the main building and at right angles to it. The ground floor of this wing is occupied by a chapel, and the upper floor is used as an isolation ward when required. Both floors are united by covered ways to the main building, these ways being freely open to the air. The buildings are kept scrupulously clean. The water supply is excellent, and the surface drains are periodically overhauled. There are no sewer connections. The milk supply is good, the cows being kept on the premises in a large, airy, well-drained byre. All milk is boiled before consumption, and the food is carefully inspected by the Sisters every day. As a general rule, the health of the inmates is excellent.

CASE I.—Kate N., aged 6, born in India; living at the European Female Orphan Asylum, Calcutta. Previous health always good. On the morning of January 17, 1894, was attacked with fever and sore throat and was put to bed. On January 18, she was seen by the medical officer of the Asylum. She had then a temperature of 104°. Her throat was sore, the fauces and tonsils being reddened. Tongue coated with a white fur with the fungiform papillæ showing through. Conjunctivæ were congested, and the skin of the neck, chest, abdomen and flexures of the elbows were covered with a bright red diffused rash, which disappeared on pressure only to return when the pressure was removed. The urine was examined and found to contain $\frac{1}{2}$ albumen on settling after boiling. Dr. William Coulter, of this city, saw the child and

pronounced the clinical characters to be those of scarlatina and advised immediate isolation. As the isolation ward was not available, arrangements were made to send the child to the European General Hospital, where she went next day. A dose of castor oil was ordered meanwhile.

For the further history of this case, we are indebted to Major H. W. Pilgrim, I.M.S., the superintendent of the hospital, who kindly placed the notes at our disposal.

January 19.—On admission to hospital, temperature 103.4, skin warm, pulse frequent. The body studded with minute spots of scarlet hue; was given a diaphoretic mixture every three hours and a gargle of boracic acid to be used frequently. The lungs, liver and spleen were pronounced normal.

January 20.—Slept well; tongue dry; pulse 120. There was an ulcer on the right tonsil and general congestion of the throat and enlargement of the cervical glands. Skin still red. The urine slightly acid, sp. gr. 1014. Albumen, *nil*. The temperature rose to 104° in the afternoon, when two grains of phenacetin were given. Diet, milk, 2 pints; chicken broth, 1 pint.

January 21.—Temperature 101° at 6 a.m. Tongue still dry; papillæ enlarged. Fauces congested; no pain on swallowing. Rash fading on the legs. Temperature 102° at 6 p.m. Tinct. aconite η . i. given with the diaphoretic mixture every three hours, and a gargle of chlorate of potash used. Took nourishment well.

January 22.—Temperature 100° at midnight. Rash fading on chest and abdomen. Throat less congested and less painful. Urine, sp. gr. 1010; acid. Albumen *nil*. Highest temperature 101°. Bowels open three times to-day. Treatment continued.

January 23.—Temperature 98.6° at 6 a.m. Rash faded everywhere. Branny desquamation on face, abdomen and sides. Complaints of itchiness. Throat much better; gland in neck smaller. No albumen found in the urine. The body was washed with 1 in 40 carbolic lotion. Temperature 101° at 6 p.m.

January 24.—Temperature 98.6° at 6 a.m., and rose only to 100° at 6 p.m. Branny desquamation of back. Congestion of throat very slight. Carbolic oil to be rubbed on the skin.

January 25.—Temperature normal, rising to 99° at 5 p.m. Skin peeling off in patches, the size of a pea. Bowels once moved. No albumen in urine. Treatment continued.

January 26.—Temperature normal all day. Desquamation continued. Glands under the chin still enlarged. Treatment continued.

January 27.—Temperature normal. Urine free from albumen. Mixture omitted.

January 28.—Temperature normal. Throat all right. Still desquamating. Diet increased. Tongue cleaning at tip only.

She continued to improve from this date. The skin of the hands peeled off on February 6. Temperature remained normal except for trifling rises to 99° on February 2 and 3. The enlargement of the tonsils slowly subsided. Desquamation continued on the hands and feet until February 18. On February 4 she was given a tonic mixture of iron and quinine and

improved steadily. She was discharged quite well on February 21.

Shortly after this patient was taken ill, three other inmates of the Asylum developed simple sore throats, without fever or rash of any kind; they were isolated for a while, and in a few days allowed to return to their everyday occupations.

On January 19, the drains of the Asylum were thoroughly overhauled by the Corporation Sanitary Authorities and pronounced in good order. The cow byre was inspected, and as several of the cows and calves were covered with an impetiginous eruption on the skin of the neck and groin, Dr. W. J. Simpson, who was then Medical Officer of Health, made cultures from the skins of these animals on January 24; but although a coccus was obtained, the results were not very definite.

The strictest enquiries were made as to possible infection from outside, but without result. Of the friends who visited the asylum, none had come in contact with any cases of fever and sore throat. No parcels had been received from Europe, and very few letters, none in fact from households where scarlatina had been treated.

CASE II.—Gertrude M., aged 13; born and bred in the plains of India; both parents European, now deceased. Brothers and sisters healthy. Has been in the Asylum five years. Previous health excellent. On the morning of June 2, 1899, she complained of feeling ill. At 6 p.m. she went to the Sister in Charge and said the roof of her mouth and throat were sore. Temperature 103°. She was at once put into the isolation ward.

On June 3, temperature ranged between 103° and 104° all day. The skin of the neck, back and arms was of a bright red colour. As the bowels were costive, castor oil was given.

June 4.—Temperature ranged from 103·6° to 105°. Bowels acted very freely. She was seen by one of us. There was a bright-red generally diffused rash all over the trunk, face and limbs, fading on pressure and returning when the pressure was removed. Fauces and uvula were acutely inflamed, as were the tonsils, which were much enlarged and covered with small patches of grey, sloughing mucous membrane. The tongue was coated white, with the fungiform papillæ showing through, giving it the typical strawberry appearance. Conjunctivæ were congested, and there was much headache. Pulse 120, and was full and bounding. Respiration, 40. Nothing abnormal was discovered in the heart, lungs, liver or spleen. Urine, sp. gr. 1024; acid, scanty, high coloured, and contained $\frac{1}{4}$ albumen on settling after boiling. A gargle of Condy was ordered, and a mixture containing chlorate of potash, quinine and perchloride of iron, to be given every three hours. Diet: milk, 2 pints; chicken soup, 1 pint.

June 5.—Temperature ranged from 103·6° to 104·6°. She slept badly. The bowels acted once scantily in the morning. Headache very severe. Rash and sore throat *in statu quo*. An aperient of Gregory's powder was given in the evening.

June 6.—Slept fitfully. Temperature varied from 102° to 104·4°. Rash fading. Fine branny desquamation observed on the chest and back, fronts of the

arms and the abdomen. She was seen by us both to-day and cultures were taken from the throat and from the desquamating skin. We were both agreed that the clinical aspect was that of true scarlatina. The patient was ordered to be soaped all over twice daily and sponged with warm phenyle and water. Mixture to be given every four hours.

June 7.—Slept better. Rash fading. Desquamation abundant. Temperature 102·7° to 104·4°. Headache less. Tongue cleaning. Throat less painful; sloughs on tonsils cleaning off. Urine acid, sp. gr. 1018, still high coloured, containing albumen a trace. Took her nourishment well. Mixture to be given every six hours. Bowels require a daily dose of Gregory's powder.

June 8.—Slept badly and woke complaining of rheumatic-like pains in the hands, feet and forearms, which got worse in the evening. These pains were relieved by fomentations. Temperature varied from 102·4° to 104°. Pulse rapid and small 118. Respiration rapid. Lungs normal and heart sounds clear. One ounce of port wine to be given daily.

June 9.—Pains in limbs very bad last night, especially in right upper arm and shoulder. Temperature 102·4° to 104°. Pulse rapid but stronger. Tongue quite clean. Desquamation beginning round fingers and toes. Throat still inflamed. To take mixture thrice daily, and two ounces of port wine in twenty-four hours.

June 10.—Slept better. Pains in limbs less. Throat much better. Temperature 101·4° to 103°. A mixture of sp. amon. arom., potass. citrat. and liq. amon. acet. to be given every four hours. Taking her food well. After to-day bowels open daily without any aperient.

June 11.—Temperature gradually coming down. Fingers tremulous. Pulse small. Respiration rapid, 52. Lungs quite clear. Urine acid, sp. gr. 1015. Albumen, *nil*.

June 13.—Temperature falling gradually day by day. Desquamation in large flakes off the dorsum of the feet. Mixture, gargle, port wine and phenyle baths continued. Appetite is improving.

June 15.—Temperature fell to normal to-day. Throat much less inflamed. Skin peeling off palms and soles. Mixture to be omitted.

June 18.—Skin peeling in large flakes off palms and soles. Right tonsil only still a little red. Urine, sp. gr. 1010; acid. Albumen, *nil*. Gargle, port wine, and baths continued. Is having chicken pish-pash and milk puddings.

June 20.—Temperature sub-normal. Still desquimating. Throat normal.

June 24.—Temperature normal. Desquamating on palms and soles and buttocks, and upper arms. Feeling stronger. Sleeps well. To have ordinary diet, and port wine one ounce only. To remain in bed and be bathed twice daily.

June 26.—We both visited the patient and took cultures from the throat and the skin. As the patient was anxious to save her hair, we recommended having it well rubbed daily with carbolic oil 10 per cent. The rest of the skin was still to be bathed with warm phenyle and water twice daily after a thorough scouring with soap. Following the advice of Dr. W. H.

Dickinson, of London, we have not oiled the skin of the body as is so often done, because in Dr. Dickinson's opinion such treatment predisposes to nephritis by hindering the action of the skin. As the patient was lying in bed, there was not much chance of scales of skin being carried about.

July 1.—Patient doing well, and still lying in bed. Desquamation had ceased except on the points of the heels. As we had obtained streptococcus from the cultures we made on the 26th ultimo, the patient was ordered to be bathed twice daily with a weak solution of creolin and hydrochloric acid.

July 4.—Cultures taken from scrapings of the skin of the right heel and back of right upper arm. Cultures also taken from right tonsil.

July 8.—Was quite well. She continued bathing twice daily for one week more and then returned to her ordinary life.

July 11.—Isolation ward disinfected with the Equifex sprayer and an acid sublimate solution. All bedding and clothes disinfected with superheated steam.

In this case we instituted most careful inquiries with a view to eliciting a history of possible outside infection. No cases have been heard of amongst the families of friends living in Calcutta, and who visit the occupants twice a month. No cases either have occurred among the troops in Fort William, Barrackpore, and Dum-Dum. Since April 1, 1899, the occupants of the Orphan Asylum have received only five letters and two parcels from the United Kingdom, and no one of these has come from a house where there has been a history of scarlatina or sore-throat. As regards communication by post with the rest of India, during the same period one parcel and one letter were received from the South Lancashire Regiment quartered at Jabalpur. Lieutenant-Colonel W. R. Quayle, I.M.S., however, wrote to us on July 1, saying no cases resembling scarlatina had been seen in the station of Jabalpur, nor among the troops quartered there.

The patient herself personally has received no letters or parcels. This second patient has remained within the Orphan Asylum compound for some months, and has had no friends come to see her from outside. However, on May 27 last, she was visited at the Asylum by two friends, a boy and a girl, aged 10 and 7 respectively. They all three played with one another, and were seen running about arm-in-arm.

These two young friends live in a large healthy house in Chowringhee and form part of a large household of European adults and children, besides the usual contingent of native servants. Careful inquiry has failed to elicit a history of sore-throat or fever in any member of this household, and the two children in question have kept perfect health for a long time past.

The cows were carefully examined by us, and nothing abnormal was discovered. We were informed that they had not been out of the compound for several months, and no new cows had been brought in by the cow-keeper.

We now give some account of our bacteriological investigation and of the experiments carried out by us in connection with the second case.

June 6, 1899.—Cultures made on agar from skin and throat of Gertrude M.

June 8, 1899.—Small colonies surrounding the particles of epidermis on the surface of the agar slope, which, when transferred by streak culture, showed a yellowish white slightly raised streak with a somewhat broken border. Smear preparations showed cocci in short chains, and clumps in which they grew in a square formation. The cultures from the throat showed two growths, one diplococcus pneumoniae of Weichselbaum; the other a coccus similar to that obtained from the skin.

June 14, 1899.—In the morning two young bull calves were inoculated in the left saphena vein on the inside of the thigh with 2 cc., each of a 20 hours' bouillon culture (1) from the throat streptococcus; (2) from the skin streptococcus. In the evening their temperatures were 104.7° and 104.8°. Both calves suffered from a distinct febrile attack lasting about five days, vide Temperature Charts. In calf No. 2, which was inoculated with the skin streptococcus, there was a general papular eruption which commenced two days after inoculation and spread over the neck, chest, flanks and abdomen. The spots were about half the size of a two-anna piece, denuded of hair and scabby. No. 1 had a few spots which came out rather later, but nothing like so general an eruptive attack. Neither of the calves had any local reaction at the seat of inoculation. They both had dry muzzles and staring coats. They lost flesh and appeared less lively than usual.

Cultures were made from the blood of both calves obtained by pricking the muzzle, but were so overgrown with a variety of bacteria that they were not persevered with. From the scaly papules on the neck of calf No. 2, which had originally been inoculated with the streptococcus obtained from the skin of the girl, a streptococcus was obtained identical in appearance with that inoculated.

June 26, 1899.—Cultures made from the throat and skin of the patient showed cocci in single and double chains, and clumps very similar to those previously obtained from the patient and the inoculated calf, though her skin had been washed twice daily with disinfectants for three weeks.

For the purpose of comparison, cultures were made from a sloughing bubo, a convalescent case of erysipelas and a suppurating carbuncle, and an assortment of septic cocci was obtained. Plate IV. shows a streptococcus obtained from the carbuncle which was the most like the coccus obtained from the patient and calf No. 2, but smaller, though cultivated for the same length of time on the same medium at the same temperature.

July 4, 1899.—Both calves were inoculated in the right saphena vein in the thigh with 2 cc. each of a twenty hours' bouillon culture of streptococcus pyogenes obtained from the carbuncle. They were both unwell after the injection, but the temperature as shown in the chart was less regular than after the former inoculation. But the most noticeable difference was that, whereas after the first inoculation neither of them had any perceptible local reaction, after the second each of them had a painful swelling at the seat of inoculation and could not put his foot to the ground. For three days they spent most of their time lying down, and if compelled to

walk went mostly on three legs. They were also much emaciated. After that the swelling gradually cleared up without suppuration.

July 4, 1899.—Cultures were again made from the throat and skin of the patient, and streptococci were obtained from both, though they were much more mixed up with other bacteria than had been the case on the former occasions.

July 4, 1899.—Cultures from throat and skin showed streptococci as before.

The fact of our getting a streptococcus in almost pure culture from the throat and skin of the patient, which on inoculation into calves produced a febrile attack of definite duration, and in one case, at least, an eruption appearing on the second day, and answering to the descriptions of Klein and Power of the so-called Hendon Disease, from which eruption a similar streptococcus was obtained, and further that this streptococcus differed from streptococcus pyogenes in the fever it produced and still more in producing no local reaction, points strongly to the conclusion that the streptococcus we obtained was the streptococcus scarlatinae and no other streptococcus, and as a corollary, that the case of the patient from whom it was derived was a case of true scarlatina and no other disease closely simulating it in the clinical symptoms. At the same time we are well aware that a single experiment is not conclusive, and consider it desirable that further experiments should be undertaken to confirm the result we obtained or prove it to have been merely a curious chain of coincidence. As a matter of speculation, we think it probable that at the time of the first case appearing in the Female Orphan Asylum the cows were suffering from Hendon Disease, though the streptococcus was not successfully isolated, and that the infection may have remained somewhere in the place ever since. The fact of no general outbreak occurring, if this hypothesis is correct, can only be attributed to the low infective power the contagium appears to possess in tropical countries.

We have carefully examined the literature on the subject of scarlatina in India with a view to giving a résumé of all cases published as having occurred in this country since 1871, and quoting the opinion of those who have written on the subject in the same period.

A case of scarlatina was reported in May, 1870, as having been seen in Simla, and with a view to placing the occurrence of true scarlatina in India in a definite position, the Editors of the *Indian Medical Gazette* addressed a circular to all medical men practising in Calcutta, calling for an expression of their opinions and a statement of their experience on the subject. A summary of the replies was published in October, 1871. As a result of their enquiries the Editors thus summarised:—"Scarlet fever does occur in India in European communities, either as an isolated case or a limited outbreak. It does not appear to show any great tendency to spread, and in all cases there is demonstration or a strong suspicion of its importation into this country from Europe. There is not a jot of evidence that the disease is indigenous or has ever occurred among the natives of the country. As far as Calcutta is concerned, the evidence now

adduced confirms our original assertion. The disease as a contagious exanthem may be said to be unknown here, and isolated instances are puzzling as to etiology. The subject is still open for discussion."

In the *Indian Medical Gazette* for May 1, 1872, Dr. Garden, of Saharanpur, related how during August and September, 1871, he attended four cases which were closely allied in their characters to scarlatina, in that sore throat, rash and desquamation were all present. In no case, however, was there albuminuria, but in one there was purulent otitis.

In the *Indian Medical Gazette* for May 1, 1876, p. 119, Dr. R. D. Murray described three cases of scarlatina, one of them fatal, treated in the Calcutta European General Hospital during December, 1875, and January, 1876. The patients, two boys and one girl, were members of a family living in Kidderpore.

CASE I.—A boy, aged 9. The rash appeared on the second day with severe tonsillitis. No albumen was found in the urine at any time. Desquamation occurred on the tenth day, and recovery ensued.

CASE II.—A boy, aged 13. Had sore-throat and an eruption on the fourth day. No albumen was found in the urine, and death took place in five days. *Post mortem*, all organs, including the kidneys, were found congested.

CASE III.—A girl, aged 5. She had sore throat and the rash appeared in forty-eight hours. The urine was albuminous. Desquamation began on the sixth day and was followed by recovery.

In the *Indian Medical Gazette*, in a letter dated February 15, 1879, it was reported that Drs. Cayley and Partridge saw a case of scarlatina in Calcutta, in a Eurasian boy, aged 12. The rash appeared on the second day. There was high fever and sore throat. The tongue had the typical strawberry appearance. There was general desquamation which came off the palms and soles in large flakes.

Dr. F. N. Macnamara, writing in 1880, in his "Climate and Diseases of Himalayan India," did not speak of scarlatina. But the same authority, when he was in practice in Calcutta, met with five cases in the children of an Armenian family living in the China Bazar.

In 1881, Professor Hirsch, of Berlin, published his Handbook of Geographical and Historical Pathology, wherein he stated that with regard to British India, some observers, such as Chevers for Bengal, Rhude for Tranquebar, Huillet for Pondicherry, the authors of reports from Madras, Collins for the plateau of the Deccan, Morehead for Bombay, and Evans for Mirzapur, declared that neither has any case of scarlatina come under their notice, nor has any such case been proved to have occurred in India at all. While others, particularly Hogg and a few practitioners in Lower Bengal, remark that the disease has often been imported into India, but has never become epidemic there, having been always limited to a few mild cases among European or Eurasian children. Professor Hirsch went on to say there are unquestionably mistakes in diagnosis underlying the statements as to the isolated occurrence of scarlatina in India, especially the mistaking of dengue for it, and considering this, Milroy comes to the conclusion that the disease is proved to have existed at one point only, viz., Colombo,

in Ceylon, and there merely to a slight extent and in a mild form.

In February and March, 1881, five cases, one of them being fatal, occurred at Rawal Pindi among the children belonging to the 8th Hussars then quartered there. Here the infection was traced to a child who arrived in the cantonment on January 25, 1881, and shortly afterwards was sent into hospital with suppurating ears followed by suppuration of the cervical glands, which ended fatally in fourteen days.

In the *Indian Medical Gazette* for March 1, 1883, Surgeon Hoey related the case of a European girl, aged 5, daughter of an officer living at Bangalore. The child was taken ill on December 3, 1882, with fever and sore throat. A rash was seen on December 4. No albumen was found in the urine. The child had the typical strawberry tongue. Desquamation began on December 8, and the temperature reached normal on December 11. The disease could be traced to no cause, the parents having received no parcels from Europe for many months.

In the *Indian Medical Gazette* for August 1, 1884, scarlatina was reported as being rather prevalent in Simla.

In 1886 Dr. Norman Chevers published his work on the Diseases of India. In this book he says he never saw any form of scarlatina in Lower Bengal, or any disease which could be fairly mistaken for it, nor had any medical man with whom he had discussed the subject met with a genuine and unmistakable case in that great province.

Maclean, in his "Diseases of Tropical Climates," published in the same year, said he never saw a genuine case of scarlatina in India. He had heard that cases of it had been seen at some of the Hill Stations of Upper India; if so, they were probably imported from England. Certainly no epidemic of scarlet fever had ever been seen in India.

In the *Transactions of the Calcutta Medical Society* for 1899 will be found a paper read by Dr. Kailas Chandra Basu, L.M.S., now Rai Bahadur, at the meeting held on April 10, 1899, entitled "Scarlet Fever in Calcutta." In 1886 there was an outbreak at 6, David Joseph's Lane, in which five children were attacked with fever, sore throat and a rash, followed by desquamation. The youngest member of the family, a child aged 12 months, was removed to a neighbouring house, but did not escape suffering slightly from the disease. A Parsi gentleman, who lived next door and was a constant visitor to the infected house, carried the infection to his own family, numbering nine souls, and all suffered from the same disease. The infection then spread northward among houses in Amratolla Lane, where, at No. 18, a child, aged 3, died in three days, and a young man living in the same house, who watched the case, contracted the disease and died in three weeks. In his case the fever did not abate, and the urine was albuminous and contained casts. In 1887, Dr. Basu saw an isolated case at 57, Cotton Street, in a boy, aged 14. In 1888 Dr. Basu observed nine cases, one of them being a woman, aged 12, who showed signs of the disease six days after her confinement, and died in three days. Her infant child lived a month and then contracted the disease, and three days later succumbed.

This woman was seen in consultation with Dr. K. McLeod, who agreed with the diagnosis. Dr. Basu summarised the symptoms in all these cases thus: (1) In each case there were sore throat, stiff neck and fever, followed by a rash on the second day. (2) There was no regularity in the rise and fall of temperature. The temperature in the majority of cases came to normal on the fifth day, and in a few cases lasted some weeks. (3) There was a desquamative stage in every case, so complete in some cases that the skin peeled off in sheets. In more than two-thirds of the cases desquamation was seen on the fifth day. (4) The eruption was always red, distinctly visible at the flexures of the joints and neck, but appeared lost in the face. Dr. Basu's patients were natives of India. The cases that occurred in Amratolla Lane were attributed to foul smelling, defective drains.

We believe that it was at the same meeting Dr. Crombie mentioned having met with a case in a European family living in Garden Reach. Here the infection was distinctly traced to a letter received from a household in England in which scarlatina had been treated.

In August, 1890, scarlatina broke out in Murree, and two mild cases occurred there among the members of the club.

At a meeting of a society held in June, 1893, at the Grant Medical College, Bombay, Lieutenant-Colonel W. K. Hatch related the history of a case occurring in a European, the disease having been contracted on board some troop ship. At the same meeting, Dr. Khory said he had met with a case in a Parsi in 1878. Dr. Dadabhai Jamsaji said he had seen two cases in European children in one house. The infection had come from Poonah, where the disease was at that time prevalent.

Sir William Moore, in the last edition of his "Family Medicine and Hygiene for India," published in 1893, p. 373, said: "Scarlet fever is not so common in India as England, but is becoming more so, consequent on successive importation."

Dr. E. A. Birch, in the third edition of his "Management and Medical Treatment of Children in India," published in 1895, p. 219, said: "Happily in India this disease is extremely rare; indeed, till late years, it was considered wholly unknown in the country, but this is not so. It is a disease requiring cold weather for its nurture; the hot season seems to stay its vitality. In the cold season it is sometimes imported with the children of European regiments, but it disappears with the increasing heat."

We have also carefully examined the reports published annually by the Sanitary Commissioner with the Government of India, with regard to the incidence of scarlatina among the European troops and followers and among the native troops and the jail population of India since the year 1882. No mention is made of scarlatina in these yearly reports until the year 1892, when a case occurred among the native troops stationed at Edwardesabad; its origin, however, does not appear to have been traced. In 1893 the European and native armies were free. In 1894 in the European army there were eight mild cases, three of which originated in England or on board ship, and five cases were untraced. In the

native army eleven cases occurred, six at Meerut, four at Tiddin and one at Fort William. In 1895 there was one untraced case among the troops at Bangalore, the native army being free. In 1896 the European army was alone affected, giving eight cases with one death. At Sitapur and Ranikhet the disease began in men fresh from home, but at Hyderabad and Quetta they were untraced. In 1897 the European army alone showed twenty-seven cases and no deaths. Fifteen cases, which were reported from Chakrata, received their infection from Meerut. The one case at Meerut was untraced. There were six untraced cases at Muttra, but the cases at Ambala and Rawal Pindi occurred in troops just arrived from England. In none of the reports was the jail population shown to be affected.

Perusal of the various reports on the Civil Medical Institutions, published by the local Governments of Bengal, Bombay, Madras, Burma, the Panjab and Hyderabad was unproductive, no cases of scarlatina having apparently been noted. We have in addition asked several of our professional acquaintances in various parts of India if they would kindly relate to us their experience with regard to the occurrence of scarlatina in India.

Dr. C. A. Fuller, who has been practising in Cawnpore for the last six years, never saw a case.

Dr. F. Van Allen, who has been practising in Madura for the past ten years, had never seen a case, though he has been constantly watching for it.

Major H. W. Pilgrim, I.M.S., had seen one case that was clinically typical of scarlatina, but he hesitated to call it so because of its being an absolutely solitary case occurring in an institution.

Dr. Baldwin Seal, who is now practising in Darjeeling, saw a couple of cases the year before last which were very like scarlatina. He also saw fourteen cases in one school this year, with sore throat and enlarged glands in the neck and with a scarlatiniform rash not typical. Temperature 103°-104°. Slight desquamation, not in the hands and feet. The whole thing was over in a fortnight, desquamation and all. None of the urine of these cases which he examined was albuminous. The glandular swelling was very marked in all cases, but no one suppurated. He added that the disease fitted in with nothing which he had seen exactly, but at the beginning the disease was more like scarlatina than anything else.

Major F. P. Maynard, I.M.S., now Civil Surgeon at Hazaribagh, formerly at Ranchi, said, that when taking over charge of a Panjab Regiment up country, a case of scarlatina in a Sepoy was handed over to him. The man was isolated in a tent, and convalescent when he saw him. He did not desquamate, and Major Maynard's opinion was against its being really scarlatina. This is the only case he ever heard of in India.

Dr. J. D. B. Fairlie, who has been practising in Colombo for the last six years, said he had never seen a case in Ceylon, and the medical men whom he had asked answered likewise. However, when he was on board the P. & O. s.s. *Oriental*, three days out of Bombay, he was called to see a European child aged 8, the son of an officer in the Army. He found the patient with a rash, rapid pulse, sore throat, coated tongue of most typical appearance and albuminuria.

He asked two officers of the Indian Medical Service to see the boy, and they concurred with him that the case was one of scarlatina. All three medical men signed a paper agreeing in the diagnosis, on the strength of which the patient was landed at Aden and taken to the bungalow of some friends, who remained practically quarantined for months from the rest of the community.

Lieutenant-Colonel G. F. A. Harris, I.M.S., had seen cases clinically indistinguishable from scarlatina in the children of a European official at Simla.

Dr. C. A. L. Faulkner, now practising at Narainganj, formerly for many years in Darjeeling, had seen but one case in Darjeeling and that was among the soldiers of the Jalapahar Depôt. The infection in this case was traced to a letter from home.

Captain R. Bird, I.M.S., had seen one case in Assam with all the clinical characters of scarlatina.

Dr. A. Powell, of Kalain, Cachar, once saw four native children in a *busti*, all of whom had fever, and tongues and throats most typical of scarlatina. The parents told him the skin had been red, but he saw no erythema himself. All the children had enlarged glands below the jaw and marked constipation. Urine was obtained from two of the children, and in one case the urine contained one-third albumen on settling after boiling; this albuminuric child had slight branny desquamation subsequently, the others had none. He diagnosed scarlatina (?). The patients lived in a *busti* some distance from his bungalow, and as the parents resented his visits, he had no control over the cases. Dr. Powell, on the whole, thinks the absence of desquamation proves they were not all scarlatina, though the throats and tongues of all were remarkably like that disease. In his letter he said that about this time he obtained plague bacilli from some cases of fever with glandular swellings in the neighbourhood.

Lieutenant-Colonel C. H. Joubert, I.M.S., said he had heard of cases occasionally in other men's practice, but had never seen one himself. An adult patient of his in the late hot weather had fever with a most marked scarlet rash from head to foot, typically like scarlatina, but without any of the confirmatory symptoms, but with subsequent desquamation, such as one gets after excessive sweating at times. Lieutenant-Colonel Joubert was sure, however, that this was not scarlatina, but heat fever in a gouty subject which almost ran to heat apoplexy. He added that, if an adult can get such an attack, why not a child. He had always thought that if the disease existed out here, one could not fail to come across it in its contagious form, which he had never seen in all the twenty odd years of work he had had out here.

Major D. G. Crawford, I.M.S., had never seen a case during nineteen years' work in India in numerous Civil Stations.

Dr. H. H. Bathe, Acting Senior Medical Officer to the East Indian Railway, said that early in 1897 he was called to see a European child in Asansol, who had a rather high temperature, quick pulse, and a bright scarlet eruption all over him. His throat was sore and the papillæ of his tongue were markedly prominent. Scarlet fever was diagnosed at once, and the diagnosis was confirmed by the

remarkably free desquamation which took place as the eruption faded. That the disease was infectious was shown by two other children and the father and mother catching it, all showing the same symptoms and all desquamating freely. The baby of the family, aged six months, escaped, and Dr. Bathe believes it is not uncommon for very young infants to escape infection. None of these cases showed any signs of kidney complication. There was nothing to show where the disease originated; and owing to strict precautions, it did not spread beyond that one family.

Dr. Sidney Smith, who has been in practice in Bombay for the last thirty-eight years, never had a case of scarlatina in his own practice; but during the seventeen years that he held the post of Port Medical Officer in Bombay, he saw a few cases among the children of soldiers arriving from England in troopships; these were at once removed to the Colaba Military Hospital and he saw no more of them.

Neither Dr. T. F. Pedley nor Dr. N. N. Parakh, both of Rangoon, have seen or heard of cases that could with certainty be described as true scarlatina.

We think the evidence we have brought forward goes to prove that scarlatina has occurred in India, and that the disease as such has not been confined to the European and Eurasian communities. Owing to the want of details, it is impossible to deduce much information regarding the season of the year when scarlatina most prevails in India; our first case occurred in the cold weather and our second in the hottest of hot weather.

In the majority of instances the source of infection could be traced directly or indirectly to Europe, but there are many cases where such infection cannot be ascertained, our own cases being among that number.

It is striking how often albumen has not been found by observers. However, its presence in scarlatina is not absolutely essential.

We believe that in no instance before our second case has a systematic bacteriological investigation been carried out in India with regard to this disease, and we hope others will employ this means of establishing the diagnosis of the cases they encounter from dengue, rubeola, &c.

If Calcutta possessed a vigorous Medical Society, a committee could be formed to report on this question, but as there is no such body to take it up, we hope that the Editors of the *Indian Medical Gazette* will come forward and place the question on a sound footing by again sending out circulars as in 1871, not to the medical practitioners in Calcutta only, but to all official and non-official medical men practising in India, Burma and Ceylon, calling for individual opinions and for particulars of cases that may have been met with during their practice in the East.

The number of our experiments has not been numerous, the reason being that we had only one case to work from; but these experiments will give an outline for the course to be pursued in verifying other cases of suspected scarlatina. Should any medical man meet with any case of suspected scarlatina, we shall be only too pleased to carry out for him the necessary bacteriological investigation should he not have the appliances at hand for the purpose.—*Indian Medical Gazette*, August 8, 1899.

News and Notes.

In our illustration this month we give a photograph of Pes Gigas. Among our numerous contributors there are doubtless some who have come across similar cases, and we should be glad if they will give their experience of this peculiar disease.

THE PROPAGATION OF DISEASES BY MEANS OF INSECTS.—*W. M. L. Coplin, M.D.* Manson's studies bearing on the inoculation of man by filaria-infected mosquitoes may be taken as fully admitted, and the possible dissemination of leprosy by mosquitoes is supported by the experiments of Alvarez, who permitted the mosquitoes to feast on the leprosy sores, and by suitable staining demonstrated within the mosquito organisms which could not be differentiated from the *Bacillus lepræ*. The work of Smith and Kilbourne (1892) on the Activity of the Tick in the Propagation of Texas Fever need only be mentioned, as it established a clear chain of evidence.

In support of the belief that bedbugs may disseminate disease, Denevre observed that the brother of a patient dead of tuberculosis acquired the disease from sleeping in the bed previously occupied by the deceased. Examination of the bedbugs, that were present in large numbers, showed that 60 per cent. possessed the power of infecting guinea-pigs. A fly travelling over an infected surface becomes laden with bacteria, and can infect a wound, and food, and drink. Apparently the fly has a striking fondness for the sputum of phthisical patients, and, as pointed out by Joly, infects the food, milk, &c., of others, or even exposed or neglected wounds or sores. In experiments in the laboratories insects were caused to walk over the growing culture in an agar-agar plate, and then, after intervals of varying lengths, the insects which had been kept under natural conditions were caused to walk over sterile culture plates. In this way the *Bacillus typhosus* was demonstrated ninety-six hours after infection of the insect. The experiments prove the assertion that any system of disinfection which does not destroy the material transported, and render the carrier harmless, is insufficient and unsatisfactory, and therefore undesirable.—*Philadelphia Medical Journal*, through *Pacific Medical Journal*.—*Public Health*.

Correspondence.

PINTA.

To the Editors of "The Journal of Tropical Medicine."

GENTLEMEN,—In your November issue, under Correspondence, Dr. Sandwith, Cairo, asks for a picture of Pinta. I have no picture at present on hand, but hope the following account will be of use to him.

Pinta (Spanish for blemish or spot) is an exceedingly common disease in this Carib reserve of about 2,000, including a few Creoles and Spaniards. The disease affects nearly every adult Carib and not a few children. Creoles and Spaniards are not affected to such a great extent. I have never noticed the disease in Europeans or white Spaniards. The colour varies with the colour of the skin, and the depth

and age of the growth. When the disease has died out for some time the part may be as white as this paper. Commonly the part has a deep red colour, or the margins are deep red and the centre white. *The part is always lighter than the healthy skin*, and may be a shade of grey or grey-brown without any red inflammatory reaction, just a little lighter than the normal skin. By mistaking the purplish skin for diseased patch and the diseased patch for healthy skin, text-books have entered a blue or purple variety of the disease. The patches are of all sizes and usually very irregular in outline, though I have seen a Carib with regular oval grey patches over his body giving him a very piebald appearance. Bony prominences are early affected, such as the knuckles, wrists, &c., but in time the whole of an extremity may be discoloured. The disease usually begins on a hand or foot, sometimes on the face. By examining a scraping in liq. potassæ under $\frac{1}{2}$ -inch objective, you see oval spores more pointed at one end, with fine double yellowish contour and containing a few yellow refractive granules, mycelial filaments with the spores attached terminally, and sometimes a mycelial head thick with spores. You will also see numbers of small pieces of broken filaments.

Chrysarobin, 1 dr. to 1 oz. of vaseline, is effective, but needs to be strong for the native skin.

If Dr. Sandwith needs any more information on this subject I will do what I can to supply him both with an account and pictures if necessary.

Yours very truly,

OSBORNE BROWNE, M.B.EDIN.

H.M. Colonial Medical Service.

Stann Creek,
British Honduras,
December 7, 1899.

P.S.—Scaling of the skin does not always occur. The scales are medium size. The Carib name is wallwall, meaning a surface not uniform in colour. The Creoles call it "speckles." If you will allow me, I could send "photos" and an account of this comparatively unimportant disease for the JOURNAL.—O. B.

CORRECTION.—In the article on "Trachoma and Race," by Dr. Yarr, which appeared in our October issue, the initial of Dr. Renner was wrong; it should read "Dr. W. Renner."—Ed.

Letters, Communications, &c., have been received from:—

- A.—Dr. R. E. Adamson (Labuan).
- B.—Dr. Osborne Browne (British Honduras).
- C.—Dr. F. Cantlie (Swatow).
- D.—Major R. Davis, I.M.S. (Belfast).
- E.—Lt.-Col. Ellis, R.A.M.C. (Ranikhet).
- G.—Dr. W. R. L. George (Porto Rico).
- H.—Dr. A. H. Hanley (Rathmines); Major Harold Hendley, I.M.S. (Punjaub).
- M.—Dr. A. D. Mackinnon (Zanzibar).
- P.—Dr. Plaxton (Jamaica).
- S.—Dr. A. Sampson (Fochow); Dr. J. Sandilands (New Hebrides); Dr. Henry Strachan, C.M.O. (Lagos).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.

British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

PLAGUE IN SIBERIA AND MONGOLIA, AND THE TARBAGAN (*Arctomys bobac*).

By FRANK G. CLEMOW, M.D., D.P.H.

THE existence of a centre of plague, or of a disease closely resembling plague, in the Transbaikial province of Siberia has been known for some time, but the accounts of it which have hitherto appeared in the English medical journals have been scanty, almost entirely derived from German sources, and in not a few respects inadequate and incorrect in details. So far as I am aware, the only first-hand information that has yet been published upon this remote centre of the disease has been contained in two articles published in the *Journal of General Hygiene and Legal and Practical Medicine*¹ (*Viēstnik Obshtchestvennoi Gigiēnui, Sudebnoi i Prakticheskoi Meditzinui*), the official journal of the Medical Department of the Russian Ministry of the Interior. The articles were written by Dr. Biéliavski and Dr. Riēshetnikof respectively, and the following is a summary of the information they contain.

Dr. Biéliavski states that the steppe regions of the Aksha military district, in the province of Transbaikal, are the endemic home of a disease which affects both man and animals. This province is, as its name implies, the province immediately to the east of Lake Baikal; Aksha itself lies almost on the Russo-Mongolian border, and approximately in longitude 115° East, and latitude 50° North. The principal centre of the disease is, however, said to be round Tzagan-Olui, a place which is not marked on the maps at my

disposal. This region is said to be over-run with many animals, the commonest of which is a rodent of the nature of a marmot, and locally called the *tarbagan*.¹ The full-grown animal measures about 65

THE TARBAGAN.

centimetres in length (about 26 inches), of which about 17 centimetres are accounted for by the tail. It has a thick fur, of a dull yellow colour, with darker shading on the back and snout, and round the lips and eyes.

The tarbagan builds for itself large underground dwellings, where it sleeps for the whole winter, from September to March. The people of the neighbourhood say that it is so regular in its habits that it goes to sleep every year on September 14, and wakes up on March 15! At times, however, it is as late as October 1 before the tarbagans begin to hibernate. The nomad Buriats on the Russo-Mongolian frontier, and also many of the Cossack population hunt the beasts for various reasons. The tarbagans become very fat in the summer, and their flesh, which is said to have an odour not unlike that of goose, is considered a great dainty by both Buriats and Cossacks. But the principal object in hunting them is to obtain the fat—particularly the peritoneal fat—which is used for greasing straps, harness, and other leather objects. For such purposes the fat is used in its raw state. It is also employed for lighting purposes. The fat is also packed in barrels and sent to some Siberian towns, such as Nertchinsk and Strétensk, where it fetches good prices.

¹ This word has appeared in German and English journals in various forms—sarbagan, sarabagan, tarabagan and others. The spelling in the text is that given by the Russian authors, who observed the disease on the spot, and must therefore be accepted as the correct one.

¹ For April, 1895.

In some years these animals are attacked by an epizootic disease. It usually comes on in the autumn, just before the commencement of the hibernating period. The people know that if the tarbagans have not gone underground by the end of September or beginning of October, they are being attacked by the disease which they call the plague (*tchuma*¹). The symptoms are briefly as follows:—The animal becomes languid, and ceases to bark; its gait is unsteady, and under one shoulder there sometimes appears a reddish, tense swelling; if far from its hole the animal fails to find it, and easily falls a prey to its foes. It is said that if it reaches its hole the other healthy animals refuse it admittance, or if it dies inside they at once remove the body outside. Sometimes the swelling is absent or very small, and the Buriats have another test to determine whether the animal is diseased or not. They cut into the sole of one paw, and if they find the blood coagulated they consider the animal diseased, and give it to the dogs to eat. It is an interesting fact that neither dogs nor wolves—the latter of which eat up the tarbagans in immense numbers—ever contract the disease. As the wolves seem to particularly like the plague-stricken animals, it is pointed out that this may explain the comparative rarity with which the disease is transmitted to human beings. An old inhabitant of the village of Sektui stated that during the past thirty years only three persons in the Cossack population had been attacked by the disease.² The danger is perfectly understood by the people, and they are careful to avoid it.

SYMPTOMS OF "SIBERIAN" PLAGUE.

At the end of August, 1889, in the village just named, in a Cossack family of ten persons, a girl aged 16 died of the plague after three days' illness; her death was followed by that of three other members of the family, including the head of the household. Six sons remained alive and quite well. The chief symptoms in those attacked were the following:—severe fever, headache, vomiting, sometimes diarrhoea, more often constipation, and pain under the armpit or in the groin, where, in some instances, glandular swellings appeared. After the

MODE OF COMMUNICATION.

death of the first patient, a female relative, also a Cossack, took the linen from the bed and washed it, and in a few days she herself fell ill and died. In this woman's family five others also caught the disease and died, a child of five years of age being the only member of the family left alive. In addition a young Buriat, aged 10, who had visited and played with some of the sick children, sickened and died. It was ascertained on inquiry that the members of the family first attacked (whose name was Epof) were occupied in catching and skinning tarbagans, and two

years later one of them again contracted the disease, just after skinning and removing the fat from a sickly-looking animal that his dog had caught and brought home. His death was followed by that of his brother, aged 5.

At the same time, in the autumn of 1881, in the town of Aksha itself, six persons died of the disease out of a Cossack family of thirteen, and two others from another family. The first case in this group was that of a man who had crossed the frontier for business purposes, and had eaten some tarbagan flesh with some Mongolians. He sickened the day after his return home, and died three days later. One of the five other members of the family attacked was sent to the hospital, but admission was refused him, and he died in the house of a neighbour. It was in this house that the other two deaths occurred. The symptoms in every case were the same as those described above.

A SEVERE VISITATION.

In September, 1894, a group of cases occurred in another Cossack family in the village of Sektui. The head of the household fell ill on September 2, and died three days later. The illness was characterised by fever, headache, drowsiness, vomiting and diarrhoea. On September 14 the youngest son, aged 8, was attacked with the same symptoms, and in addition with pain and swelling "in the armpits and groins." After this death the whole family removed to the unoccupied house where the Epof family, mentioned above, had lived and died. Here on September 15 a son, aged 10, fell ill and died on the 18th; on the 17th the mother, aged 40, fell ill and died on the 21st; on the 19th the grandfather (father of the first patient) was attacked and died on the 22nd; on the 20th the grandmother (wife of the last) fell ill and died on the same day; on the 23rd the eldest daughter, aged 20, was attacked, and, though she recovered from the acute attack, she later went out of her mind and died on October 3.

From the time of their removal to the house of the Epofs, these unfortunate people were entirely avoided by their neighbours, who dreaded, not without good reason, the risk of infection. They did not, however, leave them to die of starvation or cold, and the manner in which they provided them with food, water and firewood recalls exactly similar incidents in the well-known story of the plague epidemic at Eyam, in Derbyshire, in 1666. The villagers brought the articles and placed them on the road at some distance from the infected house, and called to those inside to come out and fetch them. Milk and water they poured from their own vessels into others put outside by the members of the infected household. When the deaths occurred the bodies were buried by the survivors, who threw into the grave the clothes and linen of the deceased. Only the first that died was buried in a coffin; the rest were buried without coffins but in separate graves, which were afterwards filled with lime. The graves were in one corner of the cemetery, which was marked off by a wooden railing, so that the earth should never be subsequently disturbed for other interments. Sixteen days after the last death the survivors removed to the house of a

¹ The Russian word *tchuma*, while used to signify the specific disease which we call plague or bubo-plague, has also a somewhat wider sense, and is applied to some other diseases. It is the word used for cattle-plague. *Sibirskaja tchuma*, or Siberian plague, is anthrax.

² This statement is, however, directly contradicted by what follows in the text.

relation, after changing all their clothes in an out-house, burning their old clothes and putting on new ones provided by the relation. As all the precautions just described were carried out on the initiative of the villagers themselves, it is impossible to withhold one's meed of admiration for the intelligence and humanity displayed, under circumstances which have but too often led to panic and acts of the most selfish inhumanity.

ORIGIN OF THE OUTBREAK.

The origin of this outbreak of plague was easily traced. The first patient had just before his illness gone to attend a court at Tzagan-Olui. On the way there his dog caught and killed, in a very brief time, six tarbagans, which the man carried some versts and then hid under some straw by the roadside, to pick them up on his return. He returned on August 31, and was taken ill on September 2. The rapidity with which the animals were caught seemed to show that they must have been suffering from disease.

Dr. Biéliavski visited this village and others shortly after the outbreak described, and from a number of questions put to the villagers he ascertained the following facts:—(1) The disease attacks the tarbagans periodically, and it is the invariable custom when any of them are caught to cut open one paw to ascertain whether they are diseased or not (as described above). (2) In some years whole *oulouses* (or nomad villages) have been carried off by the disease. (3) Thirty years before a popular method of treating the disease was to give the patient a powder made from certain glands found near the kidneys of healthy tarbagans, which were cut out, dried and powdered, and put into black tea, which was drunk by the patient. This method is no longer practised as it is considered quite useless. The disease is regarded as incurable and extremely infectious. (4) When the malady breaks out the people leave the sick to their fate, and remove to another spot; after some time they return, bury the dead, and burn the infected huts and their contents. (These statements, it will be observed, do not quite tally with the facts detailed in regard to the outbreak at Sektui.)

In October, 1888, it is added, in a hut in an *oulous* or village called Ulza, forty versts from the village of Kulusutaia, the dead bodies of six Buriats were found. The cause of death was unknown, and Dr. Ashman, of the Aksha Hospital, and a feldscher (or hospital assistant) were sent to make autopsies on the bodies, and ascertain, if possible, the cause of their deaths. The former died immediately after his return to Aksha, and the latter was found dead in the cart in which he was making the return journey. Subsequently it was proved that the Buriats had died from the tarbagan plague.

The paper of Dr. Riéshetnikof, the second of the two papers describing this disease, relates most of the above facts with some slight and unimportant variations in detail. He also adds that the tarbagan is the marmot known to science as the *Arctomys bobac*. These animals, he states, abound throughout Northern Mongolia. Their underground dwellings are of great size, and often contain families of from two to eight

members. So numerous are they that in some parts they are met with scattered over the steppe at distances of only one or two hundred yards from each other. The epizootic, it is stated, usually breaks out in years of great drought. Neither wolves, hares, dogs, eagles, nor reindeer—all of which eat up the flesh of the dead animals—are affected by the disease; nor are any domestic animals liable to contract it.

Dr. Riéshetnikof had an opportunity of observing the disease in the cases in Aksha, mentioned above, and also in the medical man and feldscher who caught the disease and died from it. He describes the symptoms as consisting of high fever, giddiness, and severe headache; the face becomes red, there is a feverish, anxious expression; the pulse becomes very rapid and progressively weaker. Some patients complained of oppression and pain in the chest, with occasional dry cough, and the expectoration of a small amount of sputum which was occasionally blood-stained. There was extreme weakness and depression, but usually the patients retained consciousness more or less to the end. There would seem to be some uncertainty about the frequency with which glandular swellings develop. Both the medical man and feldscher who died of the disease had a red and painful glandular swelling in one axilla, in which supuration did not occur. In one of the other Aksha patients there was a swelling of the inguinal glands, and it is noteworthy that Dr. Utkin, who attended the case, compared the disease with "the bubonic plague of India." In three fatal cases observed by Dr. Riéshetnikof, on the other hand, there were no glandular swellings; and he suggests that their presence may indicate the reception of the virus through the skin, their absence its reception by the alimentary canal.

No other observations upon this disease have, I believe, as yet been furnished. In the absence of bacteriological and pathological evidence, it is impossible to be certain of its nature, and it can only be asserted at present that there is, in Transbaikalia, near the Mongolian frontier, a centre of an endemic disease which, if not identical with plague, has many points in common with it.¹

PLAGUE IN EASTERN MONGOLIA.

Recently proof has been brought forward of the existence of a hitherto unknown centre of undoubted plague in Eastern Mongolia; and as the disease here is also said to be associated with an affection of the same animal—the marmot—the fact lends colour to the view that the disease just described as endemic in Transbaikalia is also plague. It has, however, to be pointed out that, whereas in the Siberian province the disease is apparently directly and solely produced in man by contact with an infected animal and in no other way, this is far from being the case in the Mongolian centre. Indeed, in the latter, the only evidence that the malady can and does affect the marmot or tarbagan is the statement of members of a Belgian mission that these animals are sometimes

¹ The most important point in which this disease appears to differ from plague is in the absence of marked mental symptoms. The description of the pulmonary symptoms resembles to some extent that of the symptoms of plague pneumonia.

attacked with sickness at the same time as human beings. It will be of interest to record what is known of this Mongolian centre of plague.¹

At a distance of twelve days' ride on horseback to the north-east of Pekin, at the foot of the great Mongolian plateau, is the small village of Toug-kia-yng-tze. It is situated in latitude 42° 3' north, and in longitude 120° east (approximately), in the midst of a small valley, and some 3,700 feet above sea level. The valley is called the valley of So-len-ko;² it is some ten miles long, and at its widest only about two hundred yards in width. It has a bend about its middle, and the village named is built just at the bend, while other villages are scattered about the neighbourhood. The valley is said to have been uninhabited twenty years ago. The soil is poor, and each year after the rains the stream running through it becomes a raging torrent. The winters are extremely cold, the thermometer at times falling to — 35° C. (— 31° F.), and this severe cold lasts for four or five months. The summer—a cold one—begins in June and ends in September.

HABITS OF NATIVES.

Many of the inhabitants are Chinese Christians from the frontier of Manchuria, who have formed a small colony in this remote district. The earth huts in which the people live are as small, close, ill-ventilated, over-crowded and filthy as the huts of all semi-civilised Eastern hill-dwellers appear to be. Typhus, small-pox, and ophthalmia are—no doubt in consequence—the three most prevalent diseases. The people themselves seem to be among the most filthy in their persons of any known race—even in China. They wash their faces once a year, their bodies never! Their clothes are worn, without change, until they drop off their bodies. They live upon rice, sorghum and a few vegetables; rice is, however, regarded as a luxury; mutton is eaten in considerable quantity. For drink they take a sort of brandy prepared from sorghum, and also tea; but tea being expensive other leaves often take its place.

No sort of measures against the spread of infectious diseases are known among these people. Infected clothes removed from the dead are at once taken into wear by the other members of the family. The dead are either buried very superficially, so that when the rains come the bodies are at once exposed, or, during an epidemic, are thrown into a neighbouring valley to be torn to pieces by wolves.

FIRST OUTBREAK OF PLAGUE.

The first appearance of plague in this neighbourhood was, so far as is known, in the autumn of 1888. It was not, however, until ten years later that it came to be heard of elsewhere, and this was due to the

action of the Belgian missionaries already referred to. It is said that in 1898 the good Fathers read a description of the epidemic in India, and at once recognised the resemblance between the disease there and that at their own doors. They consequently informed the French Legation at Pekin, who sent Dr. Matignon, the physician attached to that Legation, to study the disease on the spot. As the result of his observations the Russian Government, whose interests in that part of the world are ever increasing, and for whom the proximity of this centre of disease to the Siberian-Manchurian Railway, now in course of construction, presented an obvious danger, sent a medical mission to make further inquiries. The mission included Dr. Zabolotnui, who had already made a study of plague in India, as a member of the first Russian Plague Commission sent to Bombay in 1897, a medical student and an interpreter. They travelled overland from Russia, through Siberia and Mongolia to Pekin, and thence to the scene of the disease.

The disease, so far as could be ascertained, had been unknown here before September, 1888, when it appeared at a small village called Yan-chou-kou, lying in a valley to the north-west of Toug-kia-yng-tze, and opening into the larger valley of the So-len-ko. A girl, aged 20, who had never left the neighbourhood, was the first person attacked. The infection was imported, it was believed, by some labourers who came from some of the southern provinces of China, particularly from that of Shan-Tung. This province, it is pointed out by Dr. Matignon, is not known to contain any endemic *foyer* of plague; but he suggests that the infection might have been carried thither by sailors living on the coasts of Shan-Tung and constantly visiting Canton and Amoy, "centres of plague," and carrying clothes and other merchandise back with them. This suggestion, however, helps us no further in the desired explanation, for neither in Canton nor Amoy was plague present before the year 1894, or six years later than the appearance of the disease in the Mongolian valley in question.¹ A more plausible explanation is that the labourers "from the southern provinces" brought the infection with them from one of the centres of plague in Yunnan or Quangsi, where the disease has long been endemo-epidemic.

RECURRENCES.

Since 1888 the disease has each year reappeared in the Mongolian valley with varying degrees of severity,

¹ The fullest accounts of this centre are contained in (a) an article by Dr. J. Matignon, "La Peste Bubonique en Mongolie," (*Archives de Médecine et de Pharmacie militaires*, June, 1899, p. 463); and (b) "The Endemic Centres of Plague on the Earth's Surface and the Causes of its Spread," by Dr. D. Zabolotnui, St. Petersburg, 1899 (in Russian).

² It is apparently among the eastern slopes or offshoots of the Khingan Mountains. Dr. Zabolotnui describes it as situated "in the famous Wei-chan forest, in which the Emperor Kansu, so popular among the Chinese, used to hunt."

¹ "We can find no reliable evidence to show that the plague has been known in Canton previous to the present outbreak, although of course, from vagueness of nomenclature, the history of any epidemic in China must always be surrounded with a certain amount of doubt. Making, however, all due allowance for this, we are, after diligent inquiry, obliged to accept this statement—received alike from official, medical and lay sources—that although from time to time various epidemics have prevailed in Canton, especially in the spring of the year, the particular disease in question has not hitherto been observed." Chinese Imperial Maritime Customs Medical Reports, No. 48, "Report on the Plague prevailing in Canton during the Spring and Summer of 1894," by Alex. Rennie, M.A., M.B., C.M. Amoy was infected from Canton or Hong Kong. If plague had ever appeared in Amoy at an earlier date, I have been unable to find any mention of it in any of the Medical Reports, which extend back at least as far as 1878.

but apparently never failing. In 1896 it was particularly severe, and a detailed account of that year's epidemic is described by Dr. Matignon. It began in July, and almost simultaneously, in two houses separated from each other by a small hill, and about a mile apart. Both houses had suffered in previous years. In August it broke out in Toung-kia-yng-tze, and although a great part of the population fled, 23 deaths occurred among Christian families alone. As the Christian population was estimated at 160 souls, this mortality was as high as 14 per cent. Among the total population of all creeds it was believed that 45 deaths occurred, or 15 per cent. The case mortality was excessively high, only one patient recovering. The disease spread in August to a village called Si-Kou-Meun, a mile and a-half north of the last-named village, and in September reached Kuo-Hao, at the extreme end of the valley, where 12 deaths occurred up to November 22; to the eastward two other villages were also attacked. In all some 92 Christians, and from 160 to 180 of all creeds died in this small valley of So-len-ko—a mortality of some 13 per cent. of the total population.

In 1897 the outbreak was much milder, causing only some 13 deaths. The course followed by the disease was, as in the previous year, very erratic. The epidemic began in July, when the short summer heats were nearly over, and the nights were beginning to get cool, and after abundant rain had fallen. The symptoms observed were those of plague. The majority of patients had buboes; some had none, and these mostly presented symptoms of pneumonia. Cases with pulmonary symptoms were thought to be about one-third of the total cases.¹ It is of interest to note that the sole case in which a bubo supplicated was the case which ended in recovery. In the others death occurred before suppuration had time to develop. Dr. Zabolotnui succeeded in observing sixteen cases of the disease, and seven of these were of the pneumonic form (a proportion considerably higher than the estimate just quoted). He examined four cases bacteriologically, and obtained from them cultures of a bacillus which, on subsequent examination, proved to be a particularly virulent form of the plague bacillus.

PART PLAYED BY ANIMALS.

In regard to the infection of animals, it is noted that there was no sign of mortality among rats nor among domestic animals (Matignon). Flies were observed to die, and in 1896 the Belgian missionaries stated that they were much struck by the large numbers of dead flies seen in the rooms of people ill with the plague.² The most interesting statement, however, in this connection, is that of the Belgian Fathers, to the effect that for over ten years they had observed a disease in the marmots or tarbagans,³ which are

common in the neighbourhood. "Depuis longtemps," writes Dr. Matignon, "six ans et plus, les bons Pères voyaient des cas de peste de tarabagane parmi leurs ouailles, et ne songaient pas à mal." Unfortunately no further information is forthcoming. Neither the French nor Russian observer appears to have seen the disease in these animals. The latter only states that the animal is common in Mongolia, and that it suffers from plague in Transbaikalia (quoting the two Russian authors whose writings are summarised above). There is no evidence, one way or the other, as to the identity of the disease in man and marmot in the Mongolian centre, or of its transmission from one to the other. Certainly, were the disease solely (as in Transbaikalia) or even largely caused by contact with diseased animals, some more definite statement or direct observation would have been made by either or both the authors quoted, who personally visited the scene of the disease, and would have had facilities for seeing, or hearing of, an occurrence of the kind. It must therefore be regarded as probable that in this Mongolian centre of plague the disease is not primarily, nor to any considerable extent, due to transmission of infection from diseased marmots; and that if these animals take any share at all in the spread of the disease, they do so to an incomparably less extent than in the case of the Siberian disease.

That the tarbagan is common in Mongolia there would seem to be little doubt. The animal has been identified with the marmot known as the *Arctomys bobac*. At least six species of *Arctomys* are said to be found in various parts of Central Asia, but this would appear to be the one most widely spread. In Europe it is found as far west as Russia and eastern Germany, and in Asia its habitat is Siberia, Mongolia and Tibet. Whether it is liable to the extremely infectious and fatal disease described above, elsewhere than in the Transbaikalian province of Siberia and (possibly) the So-len-ko valley in Mongolia, is an interesting question for which, at present, no answer is possible. It would be particularly desirable to know whether the epizootic described exists or has existed at places situated between these two centres, and whether the infection has at any time been carried from one to the other by means of a chain of infected marmots. The Mongolian centre is almost due south of that in Transbaikalia, and the two are approximately some 480 miles apart.

SIBERIAN AND MONGOLIAN OUTBREAKS COMPARED.

It will be observed that these two centres possess certain points in common. They are both inhabited by a people living in an unsanitary state; they are both curiously isolated, and removed from the great centres or routes of traffic. The points of difference between the two are, however, more numerous than the points of resemblance. The Siberian centre is, apparently, in a flat steppe country; the Mongolian in a valley amongst hills, and at a considerable height above sea-level. In the former the disease occurs only in certain years, in the autumn, and solely from

¹ No autopsy could be obtained, as the Chinese never permit such a thing. The physical signs of pneumonia could not be detected in these cases, but the sputum was of pneumonic character.

² Dr. Matignon did not observe any similar mortality in flies in 1897.

³ In a note in *Janus* (November, 1899), the name of tarbagan is said to have been given to the marmot by the natives of Mongolia. According to the Russian authors quoted in the

text, the name would seem to be of Transbaikalian origin. Perhaps a skilled philologist could determine whether the word is of Buriat, Cossack or Mongolian extraction.

contact with infected tarbagans. In the latter it breaks out every year, in the summer, and quite independently (so far as the published accounts enable us to judge) of disease in the lower animals. In the former it attacks only the members of the household of the first person affected, or others who have come into immediate contact with him or them, and each outbreak is thus limited to a clearly defined group of cases; in the latter it becomes epidemic over a considerable area, it is erratic in its course, it attacks a very large proportion of the entire community in a number of separate houses and separate villages, and there is no proof, apparently, that each fresh case is due to direct contact with a preceding case. It must be confessed that in these respects the behaviour of the disease in the Mongolian centre is much more in consonance with the behaviour of plague in India and elsewhere than is that of the disease in the Siberian centre.

Before closing these notes I am desirous of calling attention to a statement which has been repeatedly made, to the effect that plague is endemic in Tibet. English, French, German and Russian writers have at some time asserted that that country is an endemic home of this disease, but in no instance that I have come across has any authority for the assertion been quoted. I have been unable to find any first-hand or reliable authority in support of the statement. Plague is endemic in the Chinese provinces bordering on Tibet, and in certain Himalayan districts in India (Kumaun and Garhwal) at no great distance from the Tibetan frontier. It has been conjectured that these two centres of the disease may find a connecting link or links in other centres of the disease in Tibet, but, so far as I am aware, the conjecture has never been supported by ascertained facts.

It is interesting in this connection to observe that the marmot, *Arctomys bobac*, is indigenous to Tibet. In the *Geographical Journal* for this month (February, 1900) are some interesting notes upon travel in Western Tibet,¹ in which the following passage occurs:—"Here, too" (beyond Kyungar) "we first made acquaintance with the Tibetan marmots, *Arctomys bobac*, called *Phutiya*, by the Bhotiyas." Then follows a brief description of their habits, and the statement that their fat is considered a specific for rheumatism. No mention is made of any disease occurring amongst them.

In conclusion, it will be seen that there are many points upon which further definite information is needed before it can be asserted that the marmot, or tarbagan (*Arctomys bobac*) does in truth play any part in the spread of the specific disease which we call the plague, or bubonic plague. Thus it is desirable that pathological and bacteriological proof should be forthcoming that the disease caused by these animals in Transbaikalia is the plague, and not some other allied disorder. More definite evidence is also wanted to prove that the disease in human beings, met with in the So-len-ko valley, which has been clearly shown to be the plague, is in any way caused

by these animals. An important link missing also is the evidence that the *Arctomys bobac*, which is widely spread in Eastern Europe, Siberia, Mongolia, and Tibet, suffers from the fatal and extremely infectious disease described above, elsewhere than in the Transbaikal province and (possibly) in the neighbourhood of the So-len-ko valley in Mongolia. Finally, it would appear that proof is wanting that either true plague, or the disease allied to it, which is caught from infected marmots, exists in Tibet.

AN UNDESCRIBED FORM OF PLAGUE PNEUMONIA, WITH FIVE CASES.

By WM. C. HOSSACK, M.D.
Calcutta.

In this paper I desire to bring to notice an indefinite and obscure form of plague, which, if attention is not called to it, may readily escape the recognition of the most careful and experienced physician. The ordinary classical form of plague pneumonia is known to all; its clearly marked physical signs, its rapid onset and course, its initial gravity, the severity of its general symptoms, and its rapidly fatal termination, render its diagnosis all too easy. Its character, in short, is fulminant.

But there is a plague pneumonia which presents quite a different clinical character, quite another combination of general and local symptoms. Its onset is not fulminant but insidious, and at the end of some five to ten days the general symptoms may be little more marked than they would be in, say, a slight attack of simple bronchitis or of bronchitis complicated by a trace of broncho-pneumonia, while the local lung symptoms may be so slight and vague that they are with difficulty recognised at all. The breathing may be little if at all quickened or laboured. On auscultation and percussion one makes out a few rales and ronchi, but for the first two or three days one may fail altogether to make out any definite physical signs of pneumonia. Then one gets a small patch, perhaps towards the apex, perhaps towards the base, where faint dulness is to be elicited, with a slight increase of vocal resonance and occasionally a few fine crepitant rales. There is little if any cough, and with it very slight expectoration, clear and mucoid, or occasionally faintly muco-purulent. Expectoration, at times, may be almost wholly wanting. Speech and intelligence may be unaffected throughout, or if they are, it is only in the latter stages; head symptoms are wanting; the eyes are not congested; the tongue-coating, white, may be very slight, and death when it comes, on the fifth to the tenth day of illness as a rule, is apt to be quite unexpected. In fact, it was this unexpected death that first led me to think those cases to be plague. In addition to the culmination in death, the most striking characteristic of those cases is the pulse; it is a pulse presenting grave characters quite uncalled for by the short duration of the illness or the limited amount of lung mischief, a pulse that indicates the presence of some acutely toxic condition. In almost every case I saw it was quick out of proportion to the

¹ By Lt.-General Sir Richard Strachey, R.E., G.C.S.I., F.R.S. His journey in Tibet was made in September, 1848; but the notes are now, for the first time, published.

local or general condition, and even where it was not found at the time of examination to be weak and running, its rapidity and general character indicated the probability of its quickly becoming so. This rapid feeble pulse, then, is an important diagnostic point. As a rule there are no enlarged glands, but if there are they come late in the illness and are ill-developed. Every case I have seen or heard of has ended in death.

So far only five cases have been directly under my observation, but from the results of the 20 to 30 investigations of deaths that passed through my hands every day, and from conversation with native practitioners, I am convinced that it was far from an uncommon type in the Calcutta epidemic of the early months of '99. In numerous cases of suspicious death where the history obtained was reliable, it was one of seven to ten days' fever, not particularly high; no delirium, no pain in the chest, no glandular swelling, and very slight cough with almost no expectoration. Exact figures as to the number of these cases I shall not attempt to give, for it is impossible to fix the boundary line between reliable and unreliable information, and in numerous cases some such colourless history as that above was originally given with a view to concealment, when the true history, as subsequently elicited, was that of typical acute plague, with well defined buboes.

I must plead for indulgence if the record of these cases that follow is incomplete, if clinical details that ought to have been present are absent, if the ultimate scientific test of cultures and *post mortems* is altogether wanting. So manifestly deficient is the record that I do not attempt to cast my description on systematic lines; all I can do is to call attention to this undescribed variety. I feel the more justification in doing so because the cases are such that from their nature they are not likely to be removed to hospital and so come under accurate European observation; and even were they to come under hospital observation, they would probably escape recognition as plague, owing to the false histories that would be given and the absence of that essential local knowledge, the knowledge of previous contact with plague cases, the knowledge of removal from an infected house, that knowledge so carefully withheld by relatives, that knowledge that only a District Medical Officer, constantly working in the huts and houses of the infected localities, can possibly obtain. The time and labour necessary to acquire that knowledge are the excuse put forward for the deficiencies of the records; only those who have worked in Calcutta can realise the difficulty with which even those were procured, scanty as they are.

In reply to the self-evident objection that the cases I give were not cases of plague at all, but cases of simple bronchitis or broncho-pneumonia, I can only call your attention to their clear connection with plague, the fact that they occurred in series with indisputable plague cases, and the fact that every case proved fatal. Had they not been plague there was no reason why death should have occurred when it did.

CASE I.—(Ser. No. 489, Ward II.)—Moti, F., 30, Chapkar, residing at 296-H Upper Chitpur Road. She was discovered during the course of a thorough

search in this bustee, just as she was walking out into the street to avoid observation. The search was in consequence of repeated deaths in the bustee of cases which before death were invariably concealed, and in which, after death, only vague and unsatisfactory information could be obtained. Two of the deaths were certainly plague, and the others probably so.

Condition on examination.—The patient was well nourished, and in her general condition there was little to indicate anything seriously wrong. She answered all questions quite rationally, and her speech, if a little weak, was otherwise normal. The tongue was slightly coated with white at the back and down the middle. The eyes were not injected. Temperature 101°, pulse 140, respiration 32. The pulse, though so quick, was regular and fairly full and strong; its rate was probably accelerated by the exertion of getting up and trying to get away.

Chest.—Respiration is a little laboured, but not much. Fine rales distinctly heard at both bases and a few ronchi, but no dulness on percussion can be made out. Vocal resonance perhaps a little increased. Very little cough and very little sputum. What there was is clear and mucoid. There are no enlarged glands present anywhere. This was the condition on the seventh day of illness, March 8, 1899. The diagnosis was bronchial catarrh, with probably a little broncho-pneumonia. I suspected it might be plague, but did not report it, as there was so little to go upon.

Dr. H. P. Bannerjee saw her next day and found her in much the same condition. The pulse was slower and better. He made out a little dulness at the bases. We were both surprised when the Vigilance Committee reported her death on the morning of the 11th, *i.e.*, after ten days' illness. There was no opportunity of making a culture.

Remarks.—There had been no actual plague case in this particular hut, but from what has been said it will be seen that the plague connection, though not absolutely certain, had been highly probable. If the case had been simple broncho-pneumonia there was nothing to explain her sudden death. She was a healthy, well-nourished woman; the pneumonia was very slight; there was nothing grave in the symptoms except perhaps the pulse.

CASE II.—Dulal Moni, F., aged 52. Hindu residing in a hut at 2-4, Nundaran Seni Street. The practitioners in charge of the case, Dr. N. L. Dey and Dr. N. Chatterjee, took me to see this woman on the morning of Sunday, March 12, 1899. She had then had three days' fever. She was a thin, old, grey-haired woman. Pulse 110, temperature 101°, respiration 30. Tongue showed a thin white coat all over it.

Condition of Chest.—Rales and ronchi heard distinctly over both bases. I thought I could make out a little dulness about the point of the left scapula, but it was extremely doubtful. The diagnosis was given as bronchial catarrh in an old and probably malarial subject. The other doctors failed to make out any pneumonia at all, and I could only say I had a faint suspicion of it, with nothing definite on which to state a diagnosis. No glands were detected on

examination. To the surprise of all concerned, she died the evening of the same day, *i.e.*, after three days' fever, on March 12.

Remarks.—There have been suspicious deaths all round the immediate neighbourhood of the hut, and though definite connection with previous cases is not made out, it is highly probable. The subsequent death of the grandson from plague pneumonia is still more convincing.

CASE III.—Hari Churn Sen, M., aged 8. Hindu, from hut at 2-4, Nundoram Sen's Street, grandson of Dulal Moni previously referred to.

It turned out afterwards that her grandson, living in the same hut, had been suffering from fever for four days, until on March 14, two days after death of grandmother, he was removed to the Ward Hospital, at 2, Nundoram Sen's Street. Dr. Das reports on this date as follows:—Temperature 103°. Has got slight cough. No pain in any part of the body. No glandular swelling. Is quite sensible. No delirium. Pulse rather feeble. Is under treatment of Dr. N. L. Dey and Dr. D. M. Chatterjee. Hut is tiled; room on ground floor.

When seen by myself on the 17th, *i.e.*, on the seventh day of illness, his condition was as follows:—

Lies collapsed on the bed. Respiration 60, nostrils dilated; temperature 104°; pulse 120, very weak. Low moaning cry.

In the left groin slight tenderness and raisin-like gland indistinctly felt in inguinal region. No other enlarged gland.

Lungs.—Examined only from the front. On percussion faint dulness could be made out here and there all over. It was distinct only at the right base. Auscultation gave fine moist rales at both bases. Towards the apices breathing was distinctly bronchial and vocal resonance was greatly increased. Dulness there, however, was indistinct. Though rhonchi were heard, there were no moist sounds.

He lingered on for nearly a week before he died.

CASE IV.—(Ser., No. 285, VI.)—Kanai, M., aged 32, caste Tantee, coolie, employed in molasses shed, close to 150-H Baranose Ghose's St., Ward VI.

History.—He got ill on March 15, 1899. His is the eighth case in a series of ten, all hailing from the same hut, as will be detailed below. He was discovered on March 18.

Condition on third day of illness.—Lies in bed almost quite indifferent, but answers on being spoken to loudly. Haggard, drawn look on face. Speech slightly affected, being rather thickened. The tongue is characteristic, *i.e.*, it is covered with a white coating, but the tip and edges are free from it. Respiration is 36, slightly laboured; pulse 104, regular and fairly strong.

Physical examination of Chest.—No dulness made out on percussion. On auscultation moist rales at both apices, particularly in the right lung. The rales are fine. Towards the bases the breath sounds are rather bronchial, but no moist sounds are to be heard. The bases themselves seem to be clear. Cough, if any, is very slight and occasional, as is also the expectoration.

No enlarged glands could be found. It was arranged, by means of money compensation, that

the patient should be removed to the Ward Plague Hospital, and that the hut should be evacuated. The patient was, however, surreptitiously removed to 7, Joga Mohun Saha's Lane, and died March 19, 1899, while being removed from this house to hospital by his friends.

Previous and subsequent cases from the same hut.—(The total population of the hut was not over twenty persons.) (1) No. 232, VI., Durga, M., aged 18, Kahar, died March 8, 1899, after five days' illness. (2) No. 248, VI., Mookta, F., aged 60, Brahmin, died March 13, 1899. Pneumonia began March 7, 1899. (3) No. 249, VI., Bassuntia, F., aged 11, Bindee, got ill morning of March 9, 1899, and died 1 a.m., March 10, 1899. (4) No. 249a, VI., Agnassia, F., aged 55, Brahmin, got ill March 10, 1899. Found with pneumonia March 13, 1899. Surreptitiously removed, and died in a day or two. (5) No. 275, VI., Bindsoor, M., aged 15, Kahar, got ill March 14, 1899, died March 17, 1899, with right femoral bubo, in Medical College Hospital. (6) No. 276, VI., Soomaria, sister of Agnassia, and had nursed her. Got ill March 16, 1899, and sent to Campbell Hospital, but could not be traced. (7) No. 277, VI., name not known, M., aged 9, Sankar, attacked March 16, 1899, at 12 noon, and died 9 p.m. the same day. (8) Kanai above described. (9) No. 302, VI., Dukhee, father of Kanai. Found with pneumonia and incipient right axillary bubo, March 20, 1899. Died in Ward Plague Hospital, March 22, 1899. (10) Jogoo, No. 472, VI., M., aged 8, Tantee, grandson of Dukhee. Got pneumonia April 14, 1899, and died April 19, 1899.

CASE V.—Chumroo, M., aged 40, Kurmi, 158-4, Maniktolla Street.

History.—The hut is one in which two plague deaths had occurred recently. Hari Lal Ghose died with a left inguinal bubo, March 27, 1899.

Chumroo got ill on March 24, 1899, was coolie in a grain shop.

Condition.—March 31, 1899. The man is fairly nourished. Leucoderma on the scalp, upper and lower extremities and several patches on the chest. Talked freely; speech not being at all affected. Could move about and sit up with ease. Tongue dry and coated. Temperature 102.5°, pulse 140, respiration 52. Eyes not injected.

Chest.—Bronchial breathing with increased vocal resonance is found over both bases behind. No moist sounds at bases unless for one or two doubtful rales in right side. Two inches below point of left scapula is a patch about two inches in diameter of distinct dulness on percussion with fine crepitant rales. Dulness fairly marked over bases.

Glands.—Raisin-like glands can be felt in both groins; these are not tender; probably they are of old standing.

He is being nursed by his son in a room about 4 ft. by 6 ft. by 7 ft.

He died quite suddenly the same night.

CLINICAL REPORT ON FOUR CASES OF SUSPECTED YAWS OR FRAMBOESIA.

By W. S. GRIFFITH, M.B., F.R.C.S.
Kimberley Hospital.

THE above-mentioned four cases were admitted to the Kimberley Hospital on April 7, 1898, having suffered for about five or six months previously to admission. They were a Kafir woman of about 35, called Feakey, and three of her children, Stemmer, a boy of between 5 and 6 years old, Kleinjan, a boy of about 4 or 5, and a baby of a little more than 1 year old.

Family history.—The mother stated that she had had three other children, who had died of different diseases of the nature of which she was unaware, but which had taken them off after they had somewhat grown and were no longer babies in arms. One of these children had accompanied the mother and the other three above-named to this hospital, and died the day after admission of broncho-pneumonia. A *post-mortem* examination of this case showed no other lesion. This child had no skin lesions such as the others presented.

Besides these six children, three of which survive and are affected with the disease hereinafter described, and three of which are dead of other diseases, she states that she has had one miscarriage, but cannot say when it occurred.

She states that none of these children suffered with eruptions or rashes as babies, and that she has never so suffered till the present occasion. The children had had sore eyes, however. Her husband is healthy as far as she knows, but lives away from her in a compound, being a miner.

This family history must not be taken as absolutely reliable, for Kafirs' memories are defective, and they have little regard to accuracy of fact; moreover, their ideas of time are most indefinite.

Present illness.—Feakey states further that the disease here dealt with first made its appearance in the case of the eldest child, Stemmer, some time before last Christmas, but more accurate information as to dates was not obtainable. It attacked him on the forehead first. The next to be affected with the disease was Feakey herself, on whom it appeared on the chin as a single spot, followed in a couple of weeks' time by other spots about the face. This also occurred before Christmas a little after Stemmer had been attacked. The third affected was the baby, on the side of the chin, one month after Feakey herself, the first spot appearing on the side of the chin about two weeks before Christmas. About the same time Kleinjan, the second surviving son, was affected on the forehead.

State on admission.—On admission here all four were affected with a seemingly similar disease of the skin, the eruption being of greatest extent in the case of Kleinjan, who, as above stated, was the latest affected, and least extensive in the case of Stemmer, who was affected first. None of the four presented any scar or mole on the limbs or bones such as might be ascribed to the existence of old syphilis, acquired or congenital. The mother had no ulcers about the genitals, nor had the children these, nor any affection of the corneæ or joints such as are found in congenital

syphilis. Nor was the typical flattening of the malar prominences present in any case, nor the affection of the nose which is so often found in congenital syphilis. The children were of course too young for the affection of the teeth to have developed which is connected with the name of Hutchinson. The mother had, however, a few small dry papules about the wrists, and the baby similar papules on the legs, in addition to the principal affection, but these papules were, I think, abortive attempts at the other rash.

The disease.—The main affection consisted of several broad flat greyish-brown plaques varying in size from that of a threepenny piece to that of a shilling, rising abruptly from the healthy skin to an elevation of about two millimetres, and covered with a crust which reformed rapidly after removal. These plaques were in some cases roughly circular, but where several were adjacent, their circumferences joined produced figures of different shape. Many of them were situated in the vicinity of skin, which was quite normal to all appearance except that it was very darkly stained. These dark patches of skin were sharply circumscribed and represented areas of disease which had healed. They were soft and elastic, showed their normal papillæ and hairs unaffected, and in fact, beyond the darker staining, were simply normal skin. The situation of these plaques was noticeable for its symmetry about the body, many corresponding parts on either side being affected, and also from the fact that the vicinity of the mouth, anus, and genitals seemed to be favourite parts. The axillæ were affected in one case. On closer examination of the plaques the surface was seen to be fissured, and on lifting off the crust the same appearance was seen in all cases, viz., an elevated area, red and papillated, like the surface of a raspberry, discharging a thin greyish fluid, and bleeding easily when touched. In the vicinity of moist parts the colour of the crust was more white and flabby, but the same raspberry appearance was always found beneath it.

The child last affected on whom the rash was most conspicuous had also some slight enlargement of the lymphatic glands about the body, but they were not very hard, nor very tender. The mucous membranes were not affected in any case, nor the throats ulcerated. The mother and baby came under my care and were treated with small doses of mercury; the other two under the care of my colleague, Dr. Booth, with bicarbonate of potash and quassia, and later, when they had begun to improve markedly, mercury was given them also, as it was thought its effect was more rapid.

One child, the one last affected called Kleinjan, during the course of treatment developed two acute attacks during which his temperature, normal before, became elevated to about 102° for three or four days, and he complained of pain in the bones, at first in the regions of the joints, though these were not swollen, and on the second occasion in the shafts of the bones. After the second attack the lower ends of his ulnæ were thickened somewhat, showing that the attack included some more or less acute periostitis. During the first attack the ends of the bones were tender on pressure. The attacks lasted about one week each.

The woman and the baby had a local application of chalk and water which was allowed to dry. The other two children were treated with carbolic oil locally. By the middle of May the woman and baby presented nothing besides the pigmented patches of skin above alluded to, which were already losing their pigment, and one small spot on the neck. The other two children were not quite well, but almost so, Kleinjan (the patient last affected) having only a few patches on his face, and one near the anus, and the slight thickening of his ulnæ above alluded to. The other child has one patch still in the axilla and one on the front of the neck—both places liable to much movement, which has delayed healing. These two latter children have lately been put on mercury, but healing was well advanced before this change.

Now the question of the nature of the disease arises, and may be shortly discussed. Was it syphilis, or was it yaws, or was it some other disease?

In favour of syphilis there are some arguments: the disease was symmetrical; in the case of the mother and baby papules were present; one child got periostitis; the mother had lost three other children, two from an unknown cause (the other, it will be remembered, died of broncho-pneumonia shortly after admission), and had had one miscarriage; syphilis is very common amongst the Kafirs and is well known to simulate other diseases; the children had had sore eyes; the disease yielded to mercury. These arguments, I think, are all that can be adduced in favour of syphilis, and they may be answered thus: yaws is also symmetrical in its distribution. Abortive yaw tubercles are described, which I think will account for the papules present on the mother and baby; moreover dry papules are very common on the skins of Kafirs from a variety of reasons, among which are parasites; an acute attack with pains in the bones is often present as the onset of yaws, though I do not remember to have seen periostitis described as an attendant feature; the three dead children died after the age of babyhood; the one miscarriage is not an uncommon event in the life of a woman free from syphilis; yaws yields to mercury, a fact which I did not know when I put the two patients on the drug; many children have sore eyes, and sore eyes alone are no argument for syphilis, if the affection be not iritis or interstitial keratitis, which these cases probably were not, as no trace is left.

Moreover, against these arguments may be adduced the facts that the eruption with its raspberry-like papillated surface is quite unlike any usual eruption in syphilis; that if it was an unusual eruption, it is very unlikely that it should occur in four patients all together; that symptoms of congenital syphilis were absent, and a history of a primary spot present in all the cases; that after this primary spot the eruption described developed in a week or two, and so must in all cases, if syphilitic, have been a very early secondary manifestation, much too early in fact; that if it was a secondary affection there was no sequence of eruption accompanying it, as would have been expected, nor were the throat or mucous membranes affected; that the plaques situated in moist places were the same as those situated elsewhere except for the colour of the scab; and that these plaques were not

like the usual mucous tubercles so well known in syphilis; finally, that there was no ulceration, and the spots healed without scar tissue forming. In fact the absence of most of the symptoms of syphilis, and the presence of an eruption quite unlike anything I have ever seen in an experience of syphilis among Kafirs—which owing to the working of the C. D. Act in Kimberley is by no means a small one—and the presence of this eruption in four cases simultaneously, make me quite convinced that the disease is not syphilis.

Other diseases which it might possibly be like, such as granuloma fungoides and the like, are very rare, and four cases together are most unlikely to occur; moreover granuloma fungoides is not, so far as I know, contagious, as this disease evidently was.

Other diseases consisting of sores covered by scabs are not more likely to account for these phenomena inasmuch as the removal of the scabs in this disease left no ulcer beneath, no loss of tissue, nor any surface like that beneath pustular eczematous scabs, which is raw but not raised or papillated as the surface was in these cases.

Finally, the appearance of the eruption, the course of the disease, the distribution of the spots, the age of the patients, mostly children, and most of the other considerations—except, perhaps, the occurrence of periostitis—are all such as are described in yaws as seen in the West Indies and Java, a disease originally coming from Africa and now frequently met with in more central parts. Consequently I am of opinion that these cases are genuine cases of yaws or framboesia, as far as clinical considerations go to determine their nature.

TYPHOID OR MALARIAL FEVER.

By W. E. DE KORTZ, M.B.Lond., &c.
Graaff Reinet, Cape Colony.

SOME remarks made by Dr. Sambon in a recent issue of your paper, "On the possible Causes of Sickness among British Troops in South Africa," dispose somewhat readily of a difficulty in diagnosis between, what I shall term adynamic remittent malarial fever, and typhoid fever. The clinical symptoms are in typical cases so similar as to require nothing short of a microscopic examination of the blood to determine the presence or absence of the plasmodium malaria, as the case may be. The term "typho-malarial" is to my mind a misnomer; it assumes the super-position of typhoid on malarial fever, or *vice versa*. I do not know whether in the Galeka-Gaika Campaign of 1887-88 microscopic examination of the blood was made, nor whether the bacillus of typhoid was shown to be present in the intestine or elsewhere; but it is perfectly certain that Dr. Woodward in 1861 did not know anything of the plasmodium malaria, and his very appellation of typho-malarial fever would indicate that his cases presented some of the features of both diseases. Whatever view of the matter is taken, the facts are these, that there is a disease practically extending from lat. 24 S. to lat. 32 S., that this disease is called typhoid by the majority of the practitioners,

and that this disease is denied to be typhoid by a majority of practitioners in South Africa. As regards this point, I may say that for years the public have been in the habit of calling the slow continuous remittent fever the "slepende" or dragging fever, and many were not attended for it professionally, and yet when this very disease becomes acute, with marked rise of temperature, &c., the doctor steps in and calls it typhoid. I have found the undoubted presence of the plasmodium malarial in cases which I and my fellow practitioners would, prior to this discovery, have called typhoid. Since this observation, we divide the cases into two groups; the mild continuous ones we call dynamic; the severe typhoid form, adynamic remittent fever.

I do not for a moment wish it to be inferred that real typhoid fever does not occur in South Africa. On the contrary, I have seen it myself; but what I am anxious to impress is that the endemic fever in South Africa, more prevalent in the spring and the autumn, but present all through the year, is remittent malarial fever, and not typhoid at all. To any one not practically acquainted with the clinical aspect of adynamic remittent fever, the error of diagnosis of typhoid is perfectly pardonable. The sorde-covered tongue, the semi-conscious state of the patient, the tense abdomen distended with flatus, the occasional bloody stools, nay, even occasional perforation of the gut, the continuousness of the fever, the frontal headache, sardonic expression, &c., all these signs and symptoms taken together afford a picture which is practically indistinguishable from typhoid; yet such a case as above described will show the presence of the malarial parasite in the blood, and with the exception of perforation of the gut, will probably get well under stimulants and quinine properly administered. I would, therefore, strongly advise our military medical men not to trust to the obvious diagnosis of typhoid fever in cases such as described above, but to use the microscope, and if the appearance of the blood does not throw considerable light on the matter, they will have lost nothing by the investigation. Briefly stated, the following are the chief points I have been able to observe in respect of which remittent fever differs from typhoid.

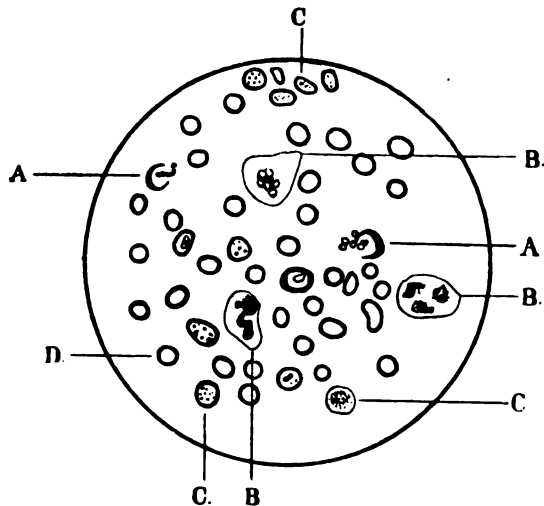
	MALARIAL REMITTENT FEVER.		TYPHOID FEVER.
	Dynamic.	Adynamic.	
Duration ..	From six days to six months		Twenty-three days, with relapses forty-eight days.
	Bilious vomiting always occurs in adynamic cases. An astonishing amount of bile is voided at times		Bilious vomiting not constant.
Tongue ..	Coating is slight; chiefly on dorsum, and middle line papillæ red at edges of tongue with fur between; somewhat like a strawberry tongue		Tongue dry, with band of white fur on each side.
	Epigastric tenderness nearly always present; splenic and hepatic tenderness nearly always present.		

Rash ..	No rose rash on abdomen; a slight roseola sometimes misleads	Rose rash.
Bowels ..	Act regularly, or no obvious fault, unless dysenteric diarrhoea complicates	Constipation well marked.
Temperature ..	At some period in twenty-four hours there is always well-marked remission, greater or lesser. Sometimes a play of 12° F. occurs between the maximal temperature in twenty-four hours	No such marked remissions occur.
Complications	Hæmorrhages, from mucous membranes, skin. Coma, hyperpyrexia, not uncommon	Thrombosis, parotitis, peritonitis, bronchitis, meningitis, orchitis.
Quinine ..	Improves all the symptoms	Quinine effect (?).
History of Infection	Is seldom obtainable; cases occur among shepherds and farmers on distant farms, where typhoid has never been known. These come in to town for medical treatment	From another case.

A possible source of infection is dead stock; it is known that among goats and sheep, as well as among oxen, epidemics of a continuous fever occur; this is considered to be malarial. In times of drought hundreds of unfortunate animals die about the homesteads. The more intelligent farmers bury the carcasses. The lazy ones do not do so, and consequently die of fever; sometimes a whole family is thus decimated. Infection from this source seems more likely when human beings drink the water from the dams at which the poor stock are watered.

The following is the method adopted for staining the blood of suspected cases of malarial fever; it is rapid and satisfactory for diagnostic purposes (*vide* Dr. Manson's "Tropical Diseases"): The forefinger is washed with soap and water, and then cleansed with absolute alcohol. Six or more microscopic slides are cleansed with absolute alcohol applied with a silk handkerchief. Cut six or more slips of cigarette paper about $\frac{1}{2}$ in. wide (a good paper to use is that known as Papier Persan); the slips are cut at right angles to the corrugations of the paper. The pulp of the finger is then pricked with 1-in. straight Hagedorn's needle, and the first drop or two of blood is wiped away and a fresh drop is exuded. The uncut edge of the slip of cigarette paper is laid on the apex of the drop, and moved gently from side to side to spread the blood; it is then lightly slid, with the blood-carrying surface downwards, on the microscopic slide. Six or more slides are thus prepared; each slide is next quickly passed through flame of spirit lamp. The film is then fixed with a few drops of absolute alcohol; four or five minutes will suffice; the alcohol is dried off with blotting paper; the film is then stained with an aqueous solution of borax methylene, 5 per cent. of the former, 2 per cent. of the latter. It is advisable to leave the stain on about two minutes. The excess of stain is washed off with water; a few minutes' exposure to the air will suffice to get rid of the water. The object is then mounted in xylol balsam, and if protected from the light the stain does not fade for several months. Other methods of staining with carbol fuchsin, eosin,

&c., are difficult to manage, and in a dry climate, owing to length of time the stain should be allowed to be in contact with the blood, the staining material is found to deposit small granules which mar the specimen. This difficulty arises from the rapid evaporation fluids undergo, and can hardly be counteracted by using moist chambers.



Stained with borax methylene; $\frac{1}{2}$ oil immersion.

A, spores bursting from a red corpuscle (due to pressure?); B, phagocytes containing sporulating bodies (?); C, red corpuscles showing stage in development of crescents of malarial parasite (æstivo-autumnal); D, normal red corpuscles.

Enclosed is a drawing of a specimen showing some red corpuscles filled with small pigmented bodies, a stage in the development of the crescent body. Two corpuscles are seen, in which the pigment bodies are bursting from the discs; this may be either due to the pressure of the cover-slip, or is the normal escape of the parasite from the red corpuscle. Several leucocytes are shown containing pigmented bodies. The specimen of blood was taken from the patient at the highest temperature point for the twenty-four hours. Temperature 103° F.

A CONTRIBUTION TO THE DIAGNOSIS AND TREATMENT OF ÆSTIVO-AUTUMNAL MALARIA.

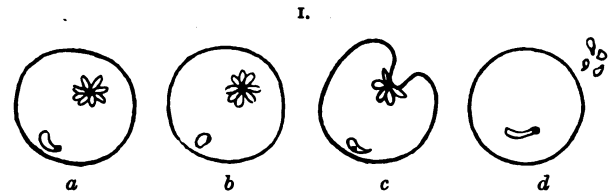
By J. PRESTON MAXWELL, M.B., F.R.C.S.
Changpoo, South China.

ANY one who has had practical acquaintance with æstivo-autumnal fever will bear me out when I assert that, for some reason or other yet unknown, quinine has neither the rapid nor the certain effect which it possesses in the treatment of the benign fevers. Æstivo-autumnal fever, when once fairly established, often resists a quinine treatment with annoying persistence and success. And it is for this reason, in part, that the case to be presently narrated is both interesting and instructive. Another reason for bringing it forward is, that it shows what information may be readily obtained by means of a good micro-

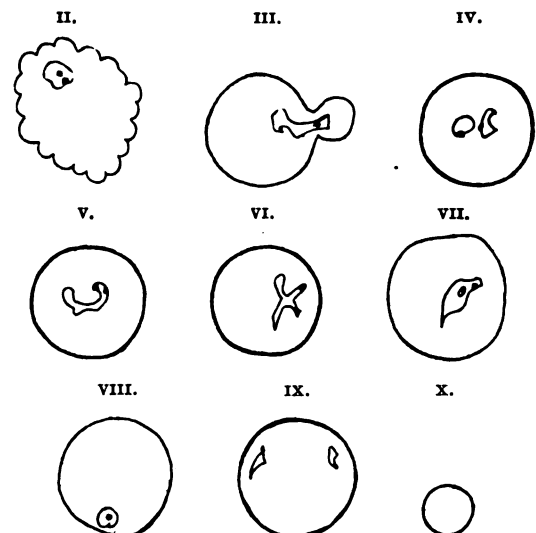
scope, and how information so acquired may be used in determining treatment.

The patient was a Chinaman, aged 26, a coolie, who had had severe previous attacks of æstivo-autumnal fever, and whose spleen was moderately enlarged. Otherwise he was in good health and full work.

On October 11, 1899, he took part in cleaning out an old foul drain, the smell from which was very bad. He remained well till October 13, and during this time the weather was dry, warm, and equable. On October 13 he came to work and worked all morning. At 1.30 p.m. he knocked off, feeling ill, and came to me at 2 p.m. to ask permission to go home and lie down.



I.—(a) Doubly infected corpuscle with sporulating parasite. Smaller actively amœboid. Pigment granules small. (c) Edge of corpuscle drawn in towards parasite. (d) Exit of spores, leaving apparently normal singly infected corpuscle.



II.—Crenated infected corpuscle.

III.—IX.—Different forms seen, all actively amœboid. In some the pigment granules were only just discernible.

X.—Colourless bodies.

No crescents visible.

Leucocytes apparently normal. No pigmented forms seen.

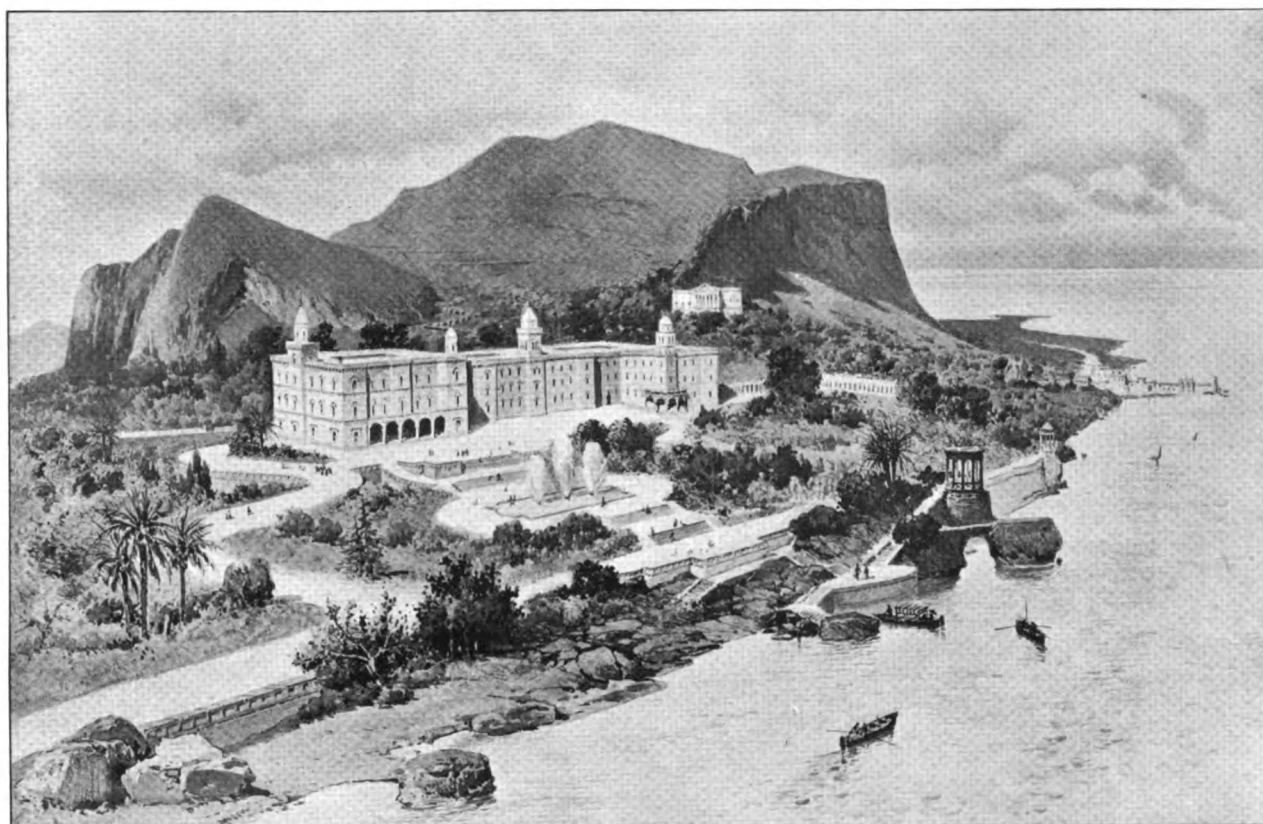
Zeiss $\frac{1}{2}$ oil immersion. Eyepiece C. (Watson's).

His condition was as follows:—He was looking wretched and pale, complaining of headache, yawning, and expressing a desire to lie down and sleep. I at once made a fresh preparation of blood, and sent him off home, giving him

Phenacetin	gr. xx.
Caffeine	gr. iv.
Pulv. ipecac. co.	gr. v.

which he took in my presence. Temperature 98.2°. Pulse 72 and quiet.

Examining the blood with an oil immersion, the presence of a parasite, which corresponded exactly



VILLA IGIEA, PALERMO, SICILY.

The Sanatorium on the shore of the Bay of Palermo with Mount Pellegrino in the background.

For description see the current issue of the Journal.

with Mannabey's *quotidian* pigmented parasite, was readily made out. In each field of the microscope there were several infected corpuscles, and some of them were doubly infected. I was fortunate enough to see sporulation in one of these doubly-infected corpuscles. There were two parasites, one of them larger than the other, with small pigment particles. The larger formed an ill-shaped rosette with tiny leaflets. After a time the surface of the corpuscle seemed drawn in towards the rosette, which became irregular and then escaped in tiny spores into the plasma. I was unable to follow the destination of these. The other parasite was actively amœboid, and was just becoming pigmented.

I went over to the patient's house at once and administered quin. sulph. gr. xv. dissolved in acid. At 7.30 p.m. he was feeling better, but very weary and still yawning. In spite of the quinine the headache was gone. I then gave him gr. v. of hydrobromate of quinine dissolved in acid into each buttock, directing the syringe fairly deeply and painting the skin with linimentum iodi. He also took another gr. x. of quinine sulphate dissolved in acid by the mouth—35 grs. in six hours, 10 being given hypodermically. His temperature was still sub-normal. He had a fair night, and at 10 a.m., the temperature not having risen, I gave him by the mouth another gr. x. of quinine sulphate dissolved in acid. He was just beginning to get a little deaf. At 2 p.m. he had a slight shivering fit, and the temperature rose to 101°4. At 4 p.m.—

Phenacetin gr. x.

Quin. sulph. gr. x.

was given, the quinine being dissolved in acid.

There were no more shivering fits, the temperature slowly descended to normal, reaching it about twenty-four hours after the first and only rigor, and has since remained normal. On the 15th and 16th quinine sulphate gr. x. in acid was given, and then all medicine was stopped. He returned to work on the 17th, his peripheral blood quite free from parasites, and has since remained well, but complained of weariness for about a week after the attack.

His own statement is that attacks beginning in this way have always been severe, and he has been twice on his back for some weeks with æstivo-autumnal fever.

I think one may fairly say, judging from the condition of the blood and the man's general state of health, that he was saved from a sharp attack of æstivo-autumnal fever by the prompt use of quinine, and it proves that in the beginning, at all events, the æstivo-autumnal parasite is in a vulnerable condition and can be successfully attacked.

NOTES FROM LAGOS, WEST AFRICA.

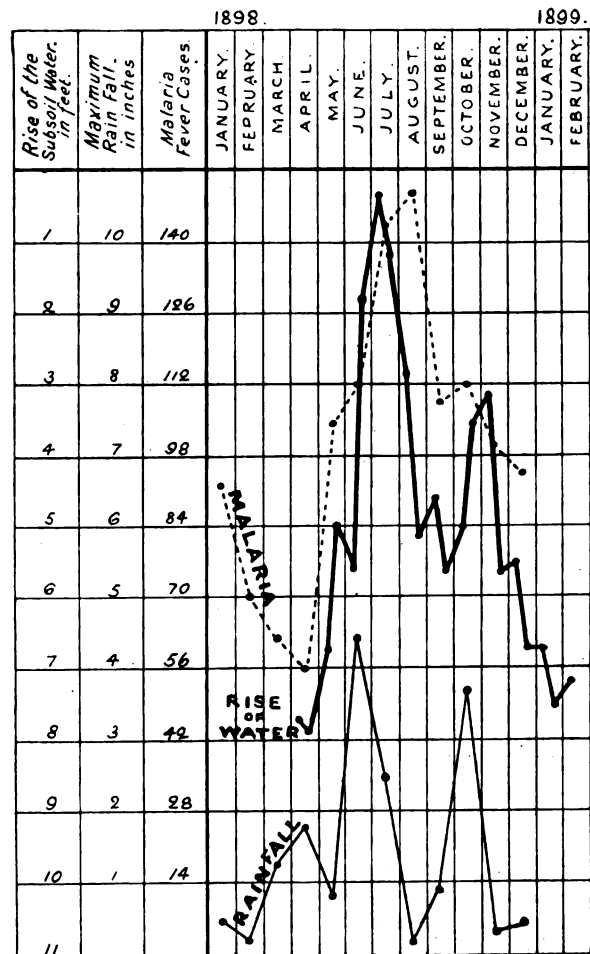
By HENRY STRACHAN, L.R.C.P.Lond., M.R.C.S.Eng., F.L.S.
Chief Medical Officer.

MALARIA, RAINFALL, AND SUB-SOIL WATER.

I HAVE for many years noted that the greatest number of cases of malarial fever occur shortly after the cessation of the rains of the tropics and but few during the actual rains, unless the latter had been interrupted by short periods of dry hot days. It

seemed, therefore, to me that saturation of the soil with water, or in other words, rise of the subsoil water, might eventually be found to have a causal relationship to malarial fever.

In Jamaica it was difficult to make any measurements bearing on the point, but the nature of the soil and position of the wells in Lagos have rendered the measuring of the distance of subsoil water from the surface easy. I was enabled, therefore, last year to carry out a series of observations in respect to the curves of rainfall, rise of ground water and malaria, which



are shown on the accompanying chart. It is interesting to note that the relationship of the malarial curve to that of the rainfall confirms the opinion expressed above, and which is shared, if I am not greatly in error, by most practitioners in the tropics. It will be seen that the height of the malarial curve follows very closely that of the ground water, and at a distance of several days the crest of the rain curve.

I think we may justly infer from this that ground saturated with water is highly favourable to the development of the malarial poison in places where that poison exists, while dry ground has the reverse action. In what way is this brought about? It would appear to be explained, having in view the mosquito hypothesis, by the fact that surface puddles

of water are formed and last longer in soil saturated with water than can possibly happen when the soil is dry. A place like Lagos, therefore, where the ground water rises to the surface and remains there for some time, should be admirably adapted as a habitat for the malarial parasite, provided that that poison and its conveyor—mosquito—be found to exist in the locality.

It is unnecessary, in view of the world-wide evil reputation as to virulent malaria attaching to Lagos, to say that the first of these conditions exists, and I have shown in the previous "Note" that mosquitos (*Anopheles*) are found here in myriads. It seems to me that entertaining either the present or the past theory of malaria in relation to wet soil a study of this chart must convince one of the extreme importance attaching to the rapid removal of ground water, and filling up of all depressions on the surface of the soil in such places as Lagos. The planting of eucalyptus, melaleuca, casuarina, cocoa-nut palm and similar trees, whose great power of extracting water from the soil is well known, should be lavishly undertaken with this object, when drainage is impossible or difficult, owing to absence of sufficient elevation above the sea level.

The number of malarial cases on the chart is the total of those appearing on the only records, namely, those of the Lagos Hospital and the Ereka Dispensary, the daily official "sick-list" and the Registrar-General's return of deaths for each month. It is obvious that a great number of cases must escape record, *e.g.*, those not medically treated, and those treated by private practitioners not ending fatally.

November, 1899.

BLACKWATER FEVER.

NUMEROUS papers have appeared on hæmoglobinuria or blackwater fever, but unfortunately none of them have cleared up the cause of the disease nor suggested the method whereby it gains entrance into the human system. Its clinical history is well known, but opinion is divided as to whether it should be regarded as a manifestation of the malarial poison or not, but there is a minority, influential and I think increasing, which regards it as a disease distinct from malaria. Until careful and exhaustive study of the blood has been made by skilled observers, this question will, I fear, remain unsettled. In the meantime there are certain facts in connection with the study of the disease which force themselves on the attention.

(1) In the malarious countries where blackwater fever is found, we know that malaria prevails at certain periods of the year, whilst at others it is comparatively absent. This is certainly not the case with blackwater fever, at least in this part of Africa. I have endeavoured in the past two years to get records of the cases, but it has been difficult to do so, and last year's record is not complete. However, I have this year, with the co-operation of some of the Government Medical Officers, been able to obtain a record of most, if not all the cases that have occurred in Lagos Colony. The result is to show that cases of blackwater fever occur all through the year, and quite independently of the presence or absence of malaria. This is well shown in the accompanying table.

TABLE SHOWING PREVALENCE OF MALARIA AND BLACKWATER FEVER IN LAGOS FROM DECEMBER, 1898, TO NOVEMBER, 1899.

	December, 1898	January, 1899	February	March	April	May	June	July	August	September	October	November	December
Malaria	94	117	90	117	92	100	212	200	141	128	102	118	..
Blackwater fever	2	1	8	—	2	1	2	3	3	2	1

(2) That quinine is not an essential in the treatment of the disease, and that the greater number of cases have been treated successfully without it, the salicylates being generally employed instead.

(3) That it appears to be a self-limited disease, which is dangerous to life from the terrible destruction of blood corpuscles which occurs whilst it runs its course.

(4) That it may attack persons who never take quinine, and that habitual quinine takers may escape it.

(5) On an investigation of the cases occurring here it is curious to note that the greater number of those attacked are Germans and Roman Catholic priests and nuns. Beyond the fact that both these classes of people do very hard work, I am unable to find that any conditions peculiar to both and different from those obtaining among the rest of the European community exist. (The theory, as to infection by some, at present unknown, living organism by means of tick bites should be borne in mind, and investigation in that direction made.)

(6) Persons already anæmic certainly appear, if attacked, to be less likely to recover, but in the twenty cases which have been noticed here in the past two years only two have died.

(7) Repeated attacks are by no means uncommon, it would almost seem that one attack predisposes to another; but more evidence on this point is wanting.

REMARKS UPON A PAPER BY DR. CALMETTE ENTITLED "INTERTROPICAL MEDICINE: ON THE PART PLAYED BY INSECTS IN THE DISSEMINATION OF THE DISEASES OF HOT COUNTRIES."

By GEORGE H. F. NUTTALL, M.D., Ph.D.
Cambridge.

THE short communication by Dr. Calmette which appeared in this Journal (January 15, p. 159) is replete with inaccuracies. In a monograph which I have recently published, Dr. Calmette and others interested in this subject will find the literature exhaustively treated.¹ From a careful perusal of the

¹ "On the *Rôle* of Insects, Arachnids, and Myriapods as Carriers in the Spread of Bacterial and Parasitic Diseases of Man and Animals: a Critical and Historical Study," *Johns Hopkins Hospital Reports*, vol. viii., October 1, 1899, 154 pages, 8 plates, and bibliography. Also in German in the *Hygienische Rundschau*, Berlin, 1899, vol. ix., the part on Malaria in the *Centralblatt für Bakteriologie*, vol. xxv. and xxvi., and in which

original literature I did not gain the impression that Davaine "on the strength of irrefutable experiments, proved the share of certain diptera in the dissemination of carbuncle." It is new to me that Magnin (Mégnin it should be) "discovered the species which enjoys this unfortunate privilege." Yersin inoculated one guinea-pig, not "Indian pigs," with the contents of a fly, not "dead flies." Dr. Calmette states that Simmond proved "that fleas are the chief agent of infection in this terrible disease." The fact is that Simmond has proved nothing of the kind, and it is altogether premature to accept Simmond's generalisations, especially when that author does not know how to distinguish one species of flea from another, and has vague ideas about the mouse-flea and *Pulex irritans* possibly being one and the same insect. The experiments of Simmond may be suggestive, but they are far too few in number to base any conclusions upon. A number of experiments made by me have given negative results. Simmond rides his hobby to death when he speculates about the possibility of plague bacilli becoming increased in virulence by passing through the body of the flea! He ignores the facts established by my experiments which proved conclusively that various pathogenic bacteria are simply digested in the alimentary canal of the flea. I by no means deny the possibility of virulent bacilli escaping in the fæces, and in fact in experiments on bugs have proved that this is the case, but only within the first twenty-four hours after they have sucked infected blood. Numerous experiments on bugs and fleas have shown that *B. pestis*, *B. anthracis*, *B. murisepticus*, and *B. cholerae gallinarum* die off in the alimentary canal of these insects. Another considerable series of experiments in which these insects were allowed to infect themselves and afterwards bite healthy animals gave uniformly negative results, proving that if this mode of infection is possible it is the exception and not the rule. Of course when an insect that has just filled itself with infected blood is crushed upon the skin, and the part scratched, infection may occur. For particulars I would refer the reader to the monograph above mentioned.

In the original publication of Spillmann and Haushalter I do not recollect the passage wherein it is written of flies "which had a predilection for settling on the lips of phthisical patients." Dr. Calmette is not only vague but inaccurate in his reference to Tictin's experiments when he writes: "The experiments of the Russian doctor are very suggestive; in opposition to the assertion of Nuttall, they prove that the germs of contagion absorbed by the bugs may preserve their virulence in spite of their contact with the digestive juices of these insects." The fact is the experiments of Tictin agree absolutely with mine. When Tictin allowed bugs to suck the blood of recurrent fever patients and immediately crushed them,

inoculating their contents into monkeys (not "various animals") he was able, as one might expect, to produce infection. A negative result was however obtained when the contents of six bugs were inoculated *forty-eight hours after* the insects had sucked blood. The spirochæte were then found to have lost their motility, though they stained normally. The fact that monkeys did not die, and that the spirilla had lost their motility, certainly indicates that these micro-organisms were on the road to dissolution. I made no "assertion" but reported exact experiments which Dr. Calmette would do well to read. Morau's experiments in the part played by bugs in the spread of "cancer" (in mice) are, he says, "so exact that they give rise to great hopes," &c. When Morau has published exact protocols of his experiments and presented evidence which can be accepted by those who are not acquainted with that gentleman, it will be time enough to dwell upon their value. I have read Morau's paper and am unconvinced. The statement, "it is known that Laveran, from the beginning of his studies, advanced the opinion that mosquitoes played the same part in the transmission of paludism as in that of filariasis," is also false. Laveran discovered the malarial parasites in 1882, and first came out as a defender of the mosquito malaria theory in 1891. I think the readers of this Journal know a little more than that the anopheles "are characterised by the presence of four white markings on the wings disposed in the form of a T." It is also new to hear that the "black spores remain in the insects, where they grow and reproduce forms capable of vegetating in the interior of the said insect." (!)

I shall not weary the reader with more criticism, but in concluding express the hope that more accurate knowledge will be gained in the future upon the part played by insects, &c., in the transmission of disease-agents. It is one thing to establish facts, it is another to write fancies. The time has passed when assertions can be accepted as evidence.

MILITARY HOSPITAL AT CAPE TOWN.—According to the Cape Town correspondent of the *Times*, complaints are being freely made about the lack of organisation which is apparent in the management of the military hospitals at Cape Town. The pressure caused by the arrival of the ambulance trains has, it is said, been too great for the staff engaged. The arrangements for the reception of the officers are held to be defective, and the number of nurses and orderlies insufficient. The statement is also made that the scale of diet allowed for serious cases is too low for healthy young officers wounded only in the extremities. We hope that in time things will shake down a bit, but there is no doubt that there is a certain amount of local jealousy in regard to the extent to which local help in the way of nursing has been declined.—*The Hospital*, January 6.

journal collective reviews of the recent progress in the investigation of malaria will continue to appear. An enlarged French edition of the above is now appearing, the translation having been made by Dr. H. Levrier (Médecin de 1ère Classe des Colonies), Bordeaux, Imprimerie du Midi, P. Cassagnol. I have a limited number of reprints of the publication in English which I shall be happy to distribute to those who are occupying themselves with investigations along these lines.

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THE

Journal of Tropical Medicine

FEBRUARY, 1900.

THE ROMAN CAMPAGNA AND MALARIA.

IN no part of the world perhaps can malaria be more efficiently or more advantageously studied than in Rome. The city itself may be pronounced as free from malaria ; yet immediately beyond the outskirts is one of the most malarial districts known. The student can make his observations and collect his facts in the infected district, and investigate and study the results of his visit to the Campagna in a city where malarial infection is practically unknown.

It is not necessary to enter into a lengthy description of this well-known district, nor is it with the whole Campagna that the study of malaria is concerned. The district geographically extends eighty-four miles from north to south, from Civita Vecchia to Terracina, and

twenty-four miles from east to west—in all an area of 1,400 square miles, with Rome practically occupying the centre.

It is with a more limited area, however, that the student of malaria has to do. The most highly malarial part of the Campagna lies in the triangular piece of land bounded by the river Tiber to the north and west, and the river Aino on the south and east. An excellent view of this flat low-lying land is to be obtained from the heights on which the town of Tivoli stands, some eighteen miles from Rome. Geologically this tract of country presents here and there lava and peperino, and the red volcanic tufa is in evidence everywhere. The surface of the land is undulating, and sluggish streams, stagnant pools and marshy land prevails. In winter the ground is covered by a coarse natural grass, with occasionally bright patches of cultivation ; during the summer, however, the winter dress disappears, and the ground becomes arid to a degree. The rivers and streams annually overflow their banks, leaving stagnant pools of water, which during the heat of summer become in many instances absolutely foetid.

It was to this district, on January 1, that a party of medical men from the London School of Tropical Medicine, consisting of Dr. Patrick Manson, C.M.G., Mr. Malcolm Morris, Dr. Sambon and Mr. Cantlie, paid a visit which was rendered both scientifically interesting and eminently authoritative, inasmuch as Professor Celli himself courteously accompanied the party and directed the observations. A number of the larvæ of the anopheles mosquito were gathered from one of the many stagnant pools, at a spot situated some five miles by railway beyond the walls of Rome. The huts in which the peasants lived were next visited, and the fact of malaria prevailing was made only too evident by the anæmic condition, the enlarged spleens, and in many instances the actual fever from which the peasants, more especially the children, were suffering. The huts in which these peasants dwell are in most instances one-room dwellings, constructed wholly of reeds. A fire of wood is made in the centre of the mud floor, and the

smoke finds exit as best it may by the doorway. The cultivation of this once fertile and populous district is reduced to a minimum; maize is the staple food of the peasants, and under existing conditions the subsistence is meagre in the extreme and but little above starvation diet.

Professor Celli has for some two years been engaged in the investigation of the sanitary problems of this malarial infected district. His observations are at once accurate, deliberate, and practical, and he has added greatly to our knowledge of the hygienic aspects of this interesting and all-important work. It is needless to say, at this late stage of our knowledge, that Celli, in harmony with Grassi, Bignami, and Bastianelli, are believers in what we term the Manson-Ross theory of malarial propagation by the mosquito anopheles. Celli has attempted, and seems to have succeeded, in establishing proof of the theory by what might be termed a negative argument. In a number of houses, principally in the signal-men's dwelling houses on the railway running through the Campagna, Celli has protected the inhabitants by mosquito nets. From May to October, when malaria is prevalent, he contrives to guard the doors and windows of dwelling-houses during the night against mosquitoes by the use of the mosquito net. In one house, that had been thus protected, the inmates consisted of a husband and wife and three children. Up to two years ago the previous occupants of this abode had all suffered from fever regularly every summer and autumn. Since then the house had been protected, fever had disappeared, and all the members of the family of the present occupants appeared in good health and condition—not quite all, as owing to the husband having to go out at night to attend to the signals in the railway he had been exposed to mosquitoes, and had had in consequence a few attacks of fever. This is at once a negative and a positive argument—negative in that those who had suffered previously were now freed of the disease, positive in that the only member of the household who was exposed suffered from fever.

An interesting fact is worthy of notice as

regards the prevalence of mosquitoes and malaria. At Acqua Albule, a hamlet on the road between Rome and Tivoli, is a thermal establishment which has acquired reputation on account of its sulphurous waters. The stream which follows through the locality is white from suspended sulphur, and gives off sulphurous acid gas in such quantity that the neighbourhood is redolent with the smell of sulphur. In this district the anopheles mosquitoes do not breed, owing, no doubt, to the sulphur in the pools. The neighbourhood has long been looked upon as a non-malarial oasis, and the beneficial influence has been ascribed to the direct action of the sulphur in the air. Whilst admitting the observation, our recent knowledge enables us to assign the freedom from malaria to the destructive action of the sulphur upon the larvæ of the mosquito anopheles. The culex, a hardier insect evidently than the anopheles, resists the sulphur fumes, and although it torments the residents by its bites, is powerless to communicate malaria.

THE JENNER INSTITUTE.

THE second series of the "Transactions of the Jenner Institute of Preventive Medicine" contains some of the results of the more important investigations which have been undertaken and pursued in the laboratories by the staff, and others engaged in research work. The contents show the Institute, under the able guidance of its director, Dr. A. Macfayden, to be gradually attaining its right position as the most important centre of research in hygiene and bacteriology in this country. No one who reads the introduction to these Transactions describing the building, laboratories and adjuncts of a truly magnificent establishment, would imagine that this Institution, of which the Council, with Lord Lister as Chairman, have reason to be proud, is the outcome of long years of struggle with difficulties, which at times, except for the unswerving perseverance and determination of those who had set their hands to it, must have proved insurmountable.

Opposition from the anti-scientists was, of course, strongly forthcoming and had to be fought against, but for many years perhaps the most discouraging difficulty was the financial one, owing to the general indifference to the important objects aimed at. Lord Iveagh's gift of £250,000 has effectually removed this source of anxiety, and now, richly endowed, the Institute will no doubt rise to the hopes and ambitions of its supporters, and Jenner's Institute will be to England what Pasteur's Institute is to France, and Koch's Institute is to Germany. The pioneers of the movement, Lord Lister, Sir Henry Roscoe, and others, are to be congratulated on their success. Initiation of a new movement in this country is particularly difficult. Conservative to a high degree and forgetful of the incessant change going on around, we are generally apt to cling to old methods adapted to a past period. This disposition has manifested itself much in the science of medicine, though in recent years it must be acknowledged there has been a change for the better in this respect; it manifests itself also in other directions, and we are now unwilling witnesses of its effects in the science of war, in which the old methods are not equal to the new. The far-reaching results in the case of war are brought very vividly and rapidly before us, but in regard to medicine and hygiene, though equally if not more far-reaching, they are not so obvious. We have eyes and we see not. Five thousand deaths a week from plague in India and 408 deaths a day in the city of Bombay in the fourth epidemic, which the inhabitants of that unfortunate town are now suffering from, are taken as a matter of course. It does not seem to occur to us that we are supremely ignorant of the means by which this disease, in its epidemic form, may be stayed or prevented, and that we can never know without systematic investigation.

Perhaps the establishment of the Jenner Institute on a firm basis will do something to assist in removing this indifference and helplessness. We should like to see some of its funds devoted to a thorough and systematic inquiry into plague from every point of view. We have long insisted on the necessity of a continuous

scientific investigation. The Plague Commission was sent out to India and has collected a mass of evidence, no doubt some of it very valuable if the important can be sifted from the unimportant, but it is not to be imagined that this can possibly take the place of a scientific investigation into plague in its different phases. And it is here that we think, if no new Commission on a scientific basis is to be despatched, that the Jenner Institute might well step in and perform a service for the country and for India, so that, in a measure, some real and sustained endeavour may be made to understand, in a better degree than is known at present, the different agents by which plague spreads, and to attempt to prevent the enormous sacrifice of life that is now going on in India.

PALERMO AS A HEALTH RESORT.

THE VILLA IGIEA.

THROUGH the public-spirited action of one of her best known citizens, the city of Palermo on the northern coast of Sicily has recently been brought prominently to notice as a health resort. Commendatore Florio, persuaded as to the suitability of Palermo as a resort for consumptives, has built a sanatorium in the immediate neighbourhood of the city, and invited a large party of medical men from this country to visit the locality and inspect the building. The party left London on December 27, and proceeded by way of Genoa, Rome and Naples to Sicily. The city of Palermo is one of the prettiest of Mediterranean towns. The population numbers over a quarter of a million people, and the busy quays, thronged harbour and imposing buildings testify to the prosperity of the inhabitants. The vegetation is sub-tropical in character and luxuriance; and during the winter the extensive orange and lemon cultivation gives the district a verdant and attractive appearance.

The climate during the winter is well nigh ideal; a bright blue sky, brilliant sunshine, still air and a pleasant temperature of between 55° and 65° allows of out-of-door life for the invalid, and for the more robust to take invigorating exercise with benefit. The city presents many attractions—a most important point in relieving the *ennui* so often attaching to invalid life spent in out-of-the-way and isolated localities. The streets of Palermo in themselves have a certain interest, and during the afternoon are thronged with the carriages of the well-to-do. There are handsome churches, excellent theatres, interesting museums, well-stocked galleries and archaeological and historical points of interest sufficient to engage the attention for several months. The amphitheatre of mountains forming the background to the city are

broken into bold peaks and ridges of imposing magnitude.

It is in the outskirts of this "Golden Shell," as the Campagna of Palermo is termed, that the sanatorium, the Villa Igia, is built—a palatial mansion, truly, both in extent and appearance. The grounds, sloping to the sea, are exquisitely laid out. Fountains, marble terraces, and a tropical wealth of flowers and palms afford pleasant paths, and in front the deep blue of the smiling Mediterranean waters complete a picture not to be surpassed, and never to be forgotten by those who have had the good fortune to witness it. Within, all that modern sanitation and Italian art can accomplish have been requisitioned to perfect this marvellous building. The impervious mosaic and concrete floors, the "prepared" walls, and the admirable system of ventilation, combine to impress the visitor with what care every detail in construction has been considered.

The bath rooms are commodious and the baths plentiful. The *cuisine* of the establishment and the attendance are of the best. Amusements of all kinds are in vogue, and ample accommodation for concerts and theatrical representations are a feature of the establishment. Last, but not least, a steam (and sail) yacht of 320 tons is placed at the disposal of visitors.

The Villa Igia can accommodate well nigh 200 guests, and both in point of size, in sanitation and in attractiveness, must be regarded as the foremost of European sanatoria.

It is difficult to find in Europe a climate and environment suitable for a change for old residents in the tropics who have taken up their abode in Britain. It is during the months of December, January, February and March that some southern resort is especially requisite. The German and French Spas are unavailable at this period, and the Riviera comes in for a large share of patronage. But the Riviera has many drawbacks: the temperature is not equable, cold snaps are frequent, and at all times an ice breeze threatens. The north of Italy is unsuitable at such a time of year, and even at Rome and Naples the weather is uncertain and variable. Palermo and the northern coast of Sicily is free from many, if not all, of these drawbacks. In addition to attractive surroundings, the climate is equable and bracing, and allows of out-of-door life in its most enjoyable and beneficial form.

We wish Commendatore Florio and all who are working with him success in their undertaking to develop Palermo as a health resort, and hope that in addition to providing a sanatorium for consumptives they may be induced to provide for the wants of a large class of persons, accustomed to tropical life, who dread the trying spring weather of Great Britain.

The party invited by Commendatore Florio included Sir Walter Foster, M.P., Professor Clifford Allbutt, Sir Lauder Brunton, Dr. Manson, C.M.G., Mr. Malcolm Morris, Dr. Gibson (Edinburgh), Dr. Sambon, Dr. St. C. Thomson, Mr. Taylor, Mr. Balestra, Dr. Norway and Mr. Cantlie.

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

RHEUMATIC FEVER IN HILL STATIONS IN INDIA.

IN the October issue of THE JOURNAL OF TROPICAL MEDICINE the question is asked as to the rarity or otherwise of rheumatic fever in the tropics. I think most army medical officers will bear me out in the opinion that this disease, which figures largely in the sick returns of the British Army in England, is comparatively rarely met with in India in the Plains. One sees cases now and then, but they are, as a rule, few and far between. This does not, however, hold good of the Hill stations in India, in which genuine rheumatic fever is as common as it is in England. In the station from which I write, I have, I think, seen more cases of rheumatic fever in six months than in fifteen years passed at various Plain stations. It is difficult to account for this prevalence, for, as far as one can see, there are no special meteorological conditions to account for it. Neither wet or cold or extremes of temperature are factors, for the cases this year have all occurred during the dry season, when the conditions were very similar to those which prevail during the more temperate months of the year in the Plains. There has, however, been another disease, not commonly met with in the Plains, prevalent at the same time, and I am disposed to think that these two may possibly stand in the relation of cause and effect. I allude to follicular tonsillitis. The connection of tonsillitis in some form with rheumatic fever has often been pointed out, but it is more as a concurrent than an antecedent affection. The following figures give the admissions for the two diseases during the eight months this station has been fully garrisoned with troops:

FOLLICULAR TONSILLITIS.

Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.
1	23	16	11	0	2	3	2

RHEUMATIC FEVER.

Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.
0	1	4	7	0	0	0	1

Two patients, both of whom had typical and severe attacks of rheumatic fever, had been in

hospital from a week to a fortnight previously with acute follicular tonsillitis, from which they had completely recovered before their re-admission for the former disease. This may be a mere coincidence, but I think it possible, had my attention been directed to the subject earlier, I might have elicited a history of a previous sore throat in others. At Aldershot, where in the days of the old wooden huts follicular tonsillitis was so common amongst the troops as to be known as the "Aldershot sore throat," rheumatic fever was extremely prevalent, and although I have no data to go upon, I am disposed to think, from my experience of that station at two separate periods, that the diminution in the cases of tonsillitis which has undoubtedly taken place since the introduction of stone barracks has been attended with an equal decline in the number of cases of rheumatic fever. At any rate, I think the coincidence of the two diseases is a matter worth investigating by those who have an opportunity of doing so, for unfortunately the Army Medical Reports throw no light on the subject.

I may add that, since it is generally admitted that follicular tonsillitis is an infectious disease, I am of opinion that the conditions which favour its prevalence in a Hill station are the existence of wooden floors and diminution in the cubic space allotted to each soldier as compared with the Plains.

P. M. ELLIS,
Lt.-Col., R.A.M.C.

Ranikhet, India.

British Medical Association.

ON FILARIAL PERIODICITY.

By PATRICK MANSON, M.D., LL.D.,
Physician to the Seamen's Hospital Society.

FILARIA BANCROFTI discharges its young (*Filaria nocturna*, the *Filaria sanguinis hominis* of Lewis) into the lymph stream. In the lymph stream the embryo parasites are carried to the general circulation, where they exhibit the peculiar phenomenon known as "filarial periodicity"—that is, they appear in countless swarms in the cutaneous circulation during the night, and disappear from it during the day.

This singular phenomenon suggests certain questions:—

I. What is the object of it?

II. Is it constant?

III. What becomes of the young filariæ during the day? (a) Do they die after a brief life of a few hours, and is there, therefore, a fresh swarm launched into the circulation every evening? or (b) do they live for many days, retiring every morning to some organ or organs, and remaining there during their daily absences from the peripheral circulation?

IV. If the latter be the case, how is it brought about?

I.

The answer to the first question, What is the object of filarial periodicity? has already been supplied. The mosquito has been shown to be an efficient intermediate host for the filaria. Bancroft in Australia has recently confirmed the original observations made in China on this point. The particular species of mosquito concerned are nocturnal in their habits. It is therefore reasonable to conclude that the nocturnal appearance of the filaria at the surface of the body is an adaptation of the habits of the parasite to those of the mosquito, its intermediate host.

II.

Is filarial periodicity constant? Yes; unless during fever, and unless the usual habits as regard the times of being awake and of sleep be inverted. In the former case periodicity is disturbed; in the latter, as shown by Stephen Mackenzie and confirmed by myself, it is correspondingly inverted.

III.

What becomes of the filaria during the day? This question a recent experience enables me to answer.

I had already ascertained by examinations of the fluids escaping in various forms of filarial lymphorrhagia that the embryo parasite is more or less constantly being passed into the lymph stream in which the parent worm lies, and that therefore filarial periodicity is not caused by a quotidian periodicity in the act of parturition. I had also ascertained, by examining the blood collected from spouting arteries severed during surgical operations performed on filariated subjects at a time when the parasite is normally absent from the cutaneous circulation, that it was present, though in inconsiderable numbers, in the smaller arteries at such time; but until recently I had no opportunity of acquiring any fairly complete conception of its exact distribution in other organs during its daily temporary absence from the surface of the body.

On February 19, 1897, a man in whose blood filaria nocturna abounded committed suicide by swallowing prussic acid. The case is recorded by Mr. Young in the *British Medical Journal*. As proved by numerous observation made at intervals during the previous nine months, this man's filariæ invariably had observed the usual quotidian periodicity, appearing in the cutaneous circulation about 5 or 6 p.m., and disappearing from it about 8 a.m. The dose of poison swallowed was apparently large. Death took place almost instantaneously about 8.30 a.m., that is just about the time when it may be assumed the filariæ had retired from the peripheral circulation for the day. The man's body was removed to Charing

Cross Hospital, and a *post-mortem* examination made the same afternoon.

Seventeen adult *Filaria Bancrofti*—16 females and 1 male—were found in an enormous lymphatic varix which occupied a great part of the pelvis and abdomen; probably many more parental worms were present, but, owing to the small size of this parasite and the difficulties of the dissection, were over-looked. At the time of the examination a hurried microscopical scrutiny of the blood from various organs gave the following result:—

A pulmonary vein contained very many embryo filariæ.

A pulmonary artery contained very many filariæ.

The margin of a lung contained prodigious numbers.

A small clot in left ventricle of heart contained a large number of filariæ.

A small clot from aorta contained a large number of filariæ.

A coronary artery and vein contained a very few filariæ.

The right ventricle of the heart contained a few filariæ.

The femoral vessels (a doubtful observation) no filariæ.

The bone marrow contained no filariæ.

The basilar artery of the brain contained two filariæ.

The middle cerebral vein contained a few filariæ.

The spleen contained no filariæ.

The liver contained no filariæ.

At the same time drops or smears of blood from the following organs were spread on microscope slips. The films were subsequently fixed with alcohol and stained with methylene blue or logwood. The filariæ on the films were then counted with the following result:—

Organ.	No. of Slides.	Aggregate No. of Filarie.	Average per Slide.
Liver	3	2	$\frac{2}{3}$
Spleen	3	3	1
Brachial venæ cœmites ..	4	111	28
Bone marrow	1	0	0
Muscle of heart	3	365	122
Carotid artery	1	612	612
Lung	10	6,751	675

The number of filariæ in the blood expressed from the lung was prodigious, as many as 30 or 40 being visible in some fields of one-sixth of an inch objective. In proportion to the amount of blood examined they were even more numerous in the thin film obtained from the carotid artery. In the lung blood they appeared to occur in groups. In the newly expressed blood most of the parasites were dead; only a few exhibited languid movements. They had the usual anatomical characters of *filaria nocturna*; when stained the sheath could readily be made out.

Through the kindness of Dr. Mott, F.R.S., pathologist to the London County Council's asylums, sections were cut from the following organs, and the filariæ, or rather fragments of filariæ (for most of the parasites were necessarily cut across in making the sections), which they contained carefully counted.

The sections were all of the same thickness and, approximately, of the same size. From three to ten sections of each organ were made, each section being from a different part of the organ. The following is the result of the count:—

Organs.	No. of Sections.	Aggregate No. of Filarie.	Average per Section.
Liver	10	3	0.3
Spleen	4	0	0.0
Kidney	8	13	1.6
Brain	4	4	1.0
Muscle (voluntary) ..	3	2	0.33
Heart muscle	4	68	17.0
Lung	6	301	50.16
Lobe of Ear	4	1	0.25
Scrotum	4	0	0.0

From these facts and figures it may be concluded that *Filaria nocturna* during its temporary absence from the cutaneous circulation is present in the larger blood-vessels, particularly the arteries; that a few are to be found in the capillaries of the muscles and brain, a few in the vessels of the kidneys, a considerable number in the muscle of the heart; but that the majority are lodged in the blood-vessels of the lungs. In the last-mentioned situation the sections showed that the filariæ are somewhat irregularly distributed, occurring in clusters in the larger vessels, or singly and more or less coiled up in the capillaries.

IV.

What is the mechanism determining this distribution of the filariæ, and what causes their absence from the cutaneous circulation during the day and presence there during the night?

This question I am unable to answer. It has been suggested that the capillaries of the skin are relaxed during sleep and so permit at that time the entrance of the filariæ. I would point out, however, that the parasites begin to come into the cutaneous circulation about 5 or 6 in the evening, and are present there in vast numbers by 9 p.m.—that is to say, long before the usual time for sleep; and that they begin gradually to diminish in number about midnight or 1 a.m.—that is, when sleep is usually profound. Therefore, although the appearance of the filariæ at the surface of the body concurs to a certain extent with the hours of sleeping, their presence there is not caused by sleep. Rather, it seems to me, their presence in the cutaneous circulation is dependent on something that tends to accumulate in the body, or in certain parts of the body, in consequence of the activities of waking life, and which tends to disappear during sleep. Manifestly, the cause is not meteorological; for, as already mentioned, the inversion of the times of waking and sleeping suffices to bring about an inversion of the periodicity.

The question suggests itself, By what means do the filariæ maintain their position in the organs they elect to occupy? How, for example, do they stem the current of blood in the aorta, or in the carotid artery, where they are in vastly greater numbers than in the slower flowing blood of the veins, as the observations I have enumerated indicate? For example, in the case of the carotid artery, the quantity

of blood which I examined was insignificant—a mere trace—and yet it contained over 600 parasites; whereas large drops from the veins contained only about 25 filariæ each. In the brain and voluntary muscles a few of the filariæ occupied the larger vessels, but the majority were in the capillaries, in which they lay lengthwise, filling the little vessels. In the lungs some were in the arterioles and outstretched, usually in company with others; many were coiled up in the capillaries and smaller vessels, the disposition of their bodies suggesting that they had assumed this particular attitude so as to jam or fix themselves in the vessel. In the kidneys they occupied principally the vessels of the Malpighian bodies; in one instance I saw three filariæ in one Malpighian tuft.

It is difficult to account for their complete absence from those highly vascular organs—the spleen, the liver, and the bone marrow.

It might be suggested that the absence of the filariæ during the day from the vessels of the skin was owing to a contracted condition of these vessels at this time. Against this supposition is the fact that there is another blood worm, *Filaria diurna*, which also observes an equally regular periodicity, but appears in the peripheral circulation during the day, and disappears from it during the night, an arrangement exactly opposite to that which obtains in the case of *Filaria nocturna*. Then there are at least two other blood worms of man—*Filaria perstans* and *Filaria demarquaii*—which are constantly present in the peripheral circulation both by day and by night. The two latter parasites are very much smaller than *Filaria nocturna*; it is therefore conceivable that they might enter vessels through which the latter could not pass.

Filaria diurna, however, is practically identical in size, shape, and structure with *Filaria nocturna*, so that what in respect to size of vessels applies to the one should equally apply to the other.

The facts seem to point to the existence of some physiological product, the outcome of the activities of waking life, which either drives *Filaria nocturna* from the surface of the body during the day, or attracts it to certain internal organs, particularly to the lungs and large arteries, during the night. What this substance may be it is for physiologists to say.

THE PERIODICITY OF THE MALARIAL PARASITE.

In conclusion, I would point to another blood parasite of man which at one period of its life is in abundant evidence in the peripheral circulation, and which at another period elects to lie up in the vessels of certain of the deeper organs. This parasite also exhibits a periodicity almost as regular as that of *Filaria nocturna*. I refer to the malarial parasite. In certain varieties of this parasite, whilst the young forms are readily obtainable in finger blood, the mature forms are almost entirely confined to the capillaries of the deeper viscera. Strange to say, the principal of these viscera are just those which are shunned by the filaria, namely, the spleen, the liver, the bone marrow, and the brain.

It is a singular and suggestive circumstance, and one which, to my way of thinking, points to some important underlying biological law, that the two

blood parasites of man subserved by the mosquito exhibit during what I might term their anthropol phase a well-marked diurnal periodicity, and, at the same time, a well-marked predilection for special but different organs during their periods of temporary absence from the peripheral circulation.—*British Medical Journal*.

SUPRAHEPATIC ABSCESS.

By JAMES CANTLIE, M.B., F.R.C.S.

Surgeon to the Seamen's Hospital Society.

DEFINITION.

By a suprahepatic abscess is meant the formation of pus between the layers of the broad ligament of the liver, having as boundaries the peritoneum (limiting the space between the layers of the broad ligament) circumferentially, the liver below and the diaphragm above. The affection may be unattended by hepatitis, dysentery, or any other abdominal ailment. It is characterised by a sudden onset, symptoms of fever, cough, and some respiratory distress and, when allowed to pursue its course spontaneously, usually terminates by the pus finding its way through the diaphragm and lung to a bronchus, from which the purulent matter is expectorated.

It will be at once evident that this not an intra-hepatic abscess; that it arises independently of any antecedent inflammation of the liver; that it may run its course without giving rise to enlargement or tenderness of the liver; and that it is independent of dysentery, although hepatitis and intestinal flux may develop during its course. In fact, it is that form of "so-called liver abscess" which arises independently of dysentery.

ETIOLOGY.

We speak of tropical liver abscess as though it were a definite disease; as though all inflammatory troubles in connection with the liver which end in pus were to be considered as constituting a definite niche in our nomenclature. This I regard as wholly unscientific, and against clinical experience and pathological evidence.

In the pathology of suprahepatic abscess dysentery plays no part, not even a secondary part, nor is it ushered in by hepatitis. The commonest cause of suprahepatic abscess is a chill. Just as acute pneumonia is consequent upon a chill in temperate climates, so is inflammation in the region I have defined a common sequel to chill in the tropics. In several cases of abscess of this nature I have met with, the disease rapidly followed a chill. A typical example will suffice.

A man, aged 28, engaged in athletic sports in Hong Kong during the month of January when the temperature in the sun is high and a rapid fall takes place when the sun goes down. He did not change his clothes after severe perspiration, and within twenty-four hours he was seized with what appeared to be sharp pleuritic pains in the lower part of the right chest. A careful examination of the lung and liver threw doubt upon the pain being due to pleurisy or hepatitis, and I diagnosed inflammatory trouble combined with effusion between the diaphragm and the

liver. I localised the situation of the lesion from previous clinical and pathological experience of similar affections and from evidence of effusion and swelling as elicited by percussion in the right suprahepatic region. As evidence of my confidence in my diagnosis, I telegraphed to the patient's brother in Formosa that his brother, under my care, had an abscess of the liver and was seriously ill. This diagnosis proved to be absolutely correct, although before the telegram was sent the patient had only been ill forty-eight hours. I mention these facts to show that it is possible to diagnose this ailment as a distinct disease from almost the onset. Pus may not have been, in all probability was not present from the beginning, but a collection of inflammatory fluid was certainly present, as evinced by the local symptoms and subsequent history. The man I speak of was an officer on a Chinese Customs cruiser, and had never had dysentery, fever, or any tropical ailment previously. This case serves as a type of many others, in which a suddenly developed suprahepatic inflammatory effusion arises, independently of dysentery or malaria, and runs a course towards suppuration. What causes the signs and symptoms to be located in this region instead of in the lungs or liver will be considered later.

ANATOMICAL CHARACTERS AND POST-MORTEM CONDITIONS.

The region to which I am calling attention is situated over that part of the liver which is destitute of peritoneum. Between the layers of peritoneum, which pass from the liver to the diaphragm, is an interval occupied by the superior vena cava, by the lymphatics passing from the upper surface of the liver towards the thorax and some small blood-vessels. The initial seat of the inflammation is in the lymphatics of this region, in the lymphatics passing from the liver to the under surface of the diaphragm. Here a lymphangitis takes place with effusion into the area, causing the liver to be pushed downwards, the diaphragm to be pressed upon from below, and the anterior and posterior aspects of the space bounded by the peritoneum to bulge. Early, very early in the disease, the local pain induces the patient to forbear using the lower part of the chest in breathing, so that the upper part of the thorax is exclusively used—a purely costal type of respiration in fact. The lower lobe of the right lung is therefore speedily congested, and pulmonary clinical signs in that region are set up. This tends to obscure the primary seat of the disease, and unless the practitioner is careful, his attention may be wholly diverted to the lung symptom—a congestion of the right base—to the neglect of the more serious condition below.

The *post-mortem* appearances, gathered from seven cases in which I had the opportunity of observing them, are as follows: On opening the abdomen, an inexperienced observer will be astounded to find that the liver, which he had regarded as the seat of the disease, and from which pus had either been withdrawn by aspiration and drainage, or been coughed up from the lung, is healthy in appearance and normal in size. He will begin to doubt, as I often did, the correctness of the diagnosis, and, believing after all

that the pus was located in the pleural cavity, proceed to open the thorax. Astonishment is followed by perplexity when, in opening the pleura, the costal and opposite visceral layer appear quite healthy, and one turns again to the abdomen in quest of explanation. There is neither pleurisy, peritonitis, nor hepatitis to account for the signs and symptoms during life. A closer inspection, however, will disclose that the anterior wall of the broad ligament seems to bulge, that on cutting it or tearing it through pus will escape, and the seat of the abscess be determined, and that the lung is adherent to the upper surface of the diaphragm by a firm basal pleurisy. An examination of the abscess cavity will show, perhaps, one, two, or more abscesses in the region. One—the large abscess—will either open upwards through the diaphragm to a bronchus, or it may have been tapped by operation during life. A second, and even a third, abscess may exist between the peritoneal layers, lying as small unburst sacs on the top of the liver. The abscesses may attain any bulk, from a hazel nut to a walnut, whilst they are unruptured. When the floor of the large abscess is examined, it is seen to be formed by the liver substance, the capsule of the liver has disappeared, and the ulcerated surface looks as if it had been scooped out of the liver quite superficially. Here an examination of the contents will reveal pus cells and broken-down red and white corpuscles in the centre, whilst around the walls will be found living white corpuscles and a few hepatic cells, and frequently the *amœba coli*. Abscess cavities which communicate with the surface, either by way of the respiratory passages or through the chest wall, may contain “infusoria” of various kinds; and in one case I found on drawing off liver pus by the aspirator what I could only consider to be empty sheaths of the *filaria nocturna*. In the unruptured abscess the pus is wholly sterile—this is the true sterile-pus abscess. Much has been written and spoken concerning the absence of pyogenic bacteria. Here, I maintain, is the solution. Abscesses the result of intestinal ulceration, be the cause of the ulceration what it may are of a septic character; but it is difficult to conceive such a process of infection arising in the situation I refer to. The pus is sterile, it is the result of a blocked lymph channel in a situation in which direct infection from without is impossible.

(To be continued.)

New Drugs and Medicines.

OXYDOL.—Practitioners in the Tropics would do well to give oxydol a fair trial in cases of dysentery, sprue, colitis, diarrhoea and enteric. Oxydol is a practically stable hydric peroxide. The oxygen, its active principle, is generated from manganese dioxide and received in water, so that an aqueous solution of oxygen is available for internal use. We have long known of the use of oxygen for inhalation and for the treatment of wounds, but never until this preparation appeared has it been possible to reach the stomach

and intestines. Nascent oxygen is liberated from oxydol during digestion, and this gas is well known to possess germicidal action of supreme power. In all intestinal ailments attended by fermentation, and therefore by pathogenic intestinal organisms, nascent oxygen in the form of oxydol is at once a safe, un-irritating and potent germicide, inhibiting fermentation, and removing thereby the chief deterrent to the cure of many of the intestinal ailments. Is constantly met with in practice in the tropics.

News and Notes.

PLAGUE AT MENGTSZ YUNNAN, CHINA.—A gentleman and lady who resided at Mengtsz, Yunnan, from June, 1896, to November, 1899, state that with the exception of 1899, plague has been epidemic in that city (population 12,000) every year for the last thirty years, or since the Mahomedan rebellion. The disease ordinarily appears about June, and finishes early in August—practically the rainy season. The annual plague mortality in the town and surrounding villages would range from 300 to 1,400. In Mengtsz rats are phenomenally abundant. Strange to say cats are very rare and are much valued, fetching high prices. The Chinese say the cats die of plague caught from the rats. As Chinese cities go, Mengtsz is clean. Every year, just before the outbreak of plague, all street rubbish is removed; domestic rubbish is stored in open places in the city and is removed at the same time. Rats dying of plague are a familiar sight. Very few women get the plague—hence the title "City of Widows" applied to Mengtsz. Possibly the comparative immunity of females depends on the careful bandaging of their feet, which in this way are protected from injury and the bites of insects. Many of the women have had several husbands, a most unusual thing in China.

Letters, Communications, &c., have been received from:—

C.—Dr. J. H. Cooke (West Indies); Dr. Carpenter (British Honduras); Dr. Tertius Clarke (Perak).
E.—Dr. F. A. Edmonds (Georgetown).
G.—Dr. W. S. Griffiths (Kimberley).
H.—Dr. W. C. Hossack (Calcutta).
M.—Dr. J. Maxwell (Highbury).
R.—Dr. Palmer Ross (British Guiana).
S.—Dr. H. Strachan (Lagos).
T.—Dr. W. Thomson (Uruguay).

EXCHANGES.

Annali di Medicina Navale.
 Archiv. für Schiffs u. Tropen Hygiene.
 Archives de Medicine Navale.
 Australasian Medical Gazette.
 Boletín de Medicina Naval.
 Boston Medical and Surgical Journal.
 Bristol Medico-Chirurgical Journal.
 British and Colonial Druggist.
 British Journal of Dermatology.
 British Medical Journal.

Climate.
 Clinical Journal.
 Clinical Review.
 Giornale Medico del R. Exercito.
 Il Policlinico.
 Indian Engineering.
 Indian Medical Gazette.
 Indian Medical Record.
 Janus.
 Journal of Balneology and Climatology.
 Journal of Laryngology and Otology.
 La Grèce Médicale.
 Lancet.
 Liverpool Medico-Chirurgical Journal.
 Medical Brief.
 Medical Missionary Journal.
 Medical Record.
 Merck's Archives.
 New York Medical Journal.
 Pacific Medical Journal.
 Polyclinic.
 Public Health.
 Revista Medica de S. Paulo.
 South African Medical Journal.
 The Hospital.
 The Medical and Surgical Review of Reviews.
 The Northumberland and Durham Medical Journal.
 Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

A CASE OF AINHUM.

By A. B. DALGETTY, M.B., C.M.
South Sylhet, India.

THE subject of this peculiar disease, which is rarely met with in India, is a Hindu washerman, 30 years of age, married, and a native of Sylhet. His parents died while he was still young, but of what diseases could not be ascertained. None of his ancestors suffered, and none of his relatives suffer, so far as he knows, from this affection.

It began about eight years ago, on the inner aspect of the root of the fifth toe of the left foot, then the fourth toe of the same foot became attacked, and now the fifth toe of the right foot shows the same process starting. (This symmetrical attack of the two little toes seems to be the rule.)

As will be seen from the photograph, the roots of the fourth and fifth toes of the left foot are encircled by a well-marked constricting furrow which is deepest on the inner aspect of each. There is no ulceration in the groove, and, beyond a slight feeling of tightness at the spot, there are no subjective symptoms. The distal ends of the toes are somewhat enlarged, are smooth in appearance, but sensation is normal, and the toes seem quite healthy.

The fifth toe of the right foot is also affected, but as the groove lies on the inner aspect of the toe the photograph does not show it so well.

The patient's general condition is fair, but I should not say robust: he shows no evident signs of syphilis or leprosy.

As to the cause of this peculiar disease nothing defi-

nite can be learned. He says that it came on of itself, he never wore toe-rings, nor had any severe injury to his toes, and there is not the least reason to suspect that he is malingering; indeed, he did not think anything about the condition till I drew his attention to it.

The only reference to ainhum in my possession is the article by Dr. Manson, in Davidson's "Diseases of Warm Climates" (pp. 993 *et seq.*). The theories there given as to its probable cause are:—Malingering, a linear scleroderma and a tropho-neurosis, but all are considered unsatisfactory explanations. The author himself inclines to the view that the fibrotic change is analogous to keloid, and is started by an injury or irritation of some sort. Might it not be worth while to consider whether it has any relation to leprosy? We sometimes see leprosy parts strangulated off by a fibrotic constriction without much ulceration, and ainhum ends by the toe being cast off. The position of the fifth toe, which almost invariably is the first to become attacked, renders it very liable to slight abrasions and cracks, into which dirt and germs of all kinds are bound to get access, owing to the natives going about barefooted. In a place where leprosy abounds the bacillus of that disease might thus easily gain a footing.

The constricting furrow is a band of fibrous tissue, and it might well be that the presence of the bacillus lepræ stimulates the connective-tissue cells to unusual activity, for, in the article on leprosy by Macnamara, in Davidson's book above referred to, occur these words (p. 441):—"Without the nourishment which the connective-tissue nucleus secretes, the leprosy bacillus cannot live; this secretion appears to be the appropriate food of this micro-organism."

The reason why the process assumes the form of an

encircling band may be solely due to some local anatomical arrangement of the subcutaneous connective-tissue.

It will be interesting to watch the further development of this case. In addition to this disease the patient suffers also from a well-marked keratosis of the palms of the hands and soles of the feet. The skin on these parts is raised, thickened, hard and horny to the touch, and worm-eaten or pitted in appearance.

It causes stiffness and want of pliancy of his fingers and hands, which interferes with movement. This keratosis began about the same time as the ainhum began, and seems to be getting worse. He also suffers from ringworm of the nails.

I am indebted to Dr. Powell, of Cachar, for kindly taking and sending me photographs of the case.

FIVE CASES OF TERMINAL DYSENTERY.

By W. J. BUCHANAN, B.A., M.B., Major, I.M.S.
Superintendent, Central Prison, Bhagalpur, Bengal.

At the meeting of the British Medical Association, in July, 1899, there was read for me a paper on what I called "Terminal Dysentery," giving notes of twenty-eight cases in which I had observed the phenomenon. I herewith publish five more cases, which I have met with since the former paper was written.

It appears certain that the occurrence of dysentery as a terminal symptom of many diseases of the tropics, is a fact. In the discussion at the Tropical Section, Professor Kenneth Macleod mentioned that he had frequently observed dysentery as a precursor of death in Leper and Lunatic Asylums in India, and Dr. Sandwith, of Cairo, has observed it in the last stages of cases of pellagra in Egypt. My cases showed that it occurred in the last stages of numerous diseases in India, in tertiary syphilis, in tuberculosis, in advanced malarial cachexia—in fact, in any lingering, wasting disease. In the discussion alluded to Dr. Manson and Dr. Sambon explained the occurrence of dysentery in these cases on the view that, owing to the debilitated state of the patients, the omnipresent germs of dysentery or the previously latent germs of dysentery in the patient's intestine became capable of producing their specific effects. This is an easy explanation, but it does not explain how it is that many other patients living under identical conditions and in the same ward, and suffering from the same diseases, escape. In addition to the five cases given below I have had at least four similar cases which proved fatal from the original disease, in whom I fully expected to find this phenomenon, but it did not occur, and in the *post-mortem* register I have specially noted that there was "no terminal dysentery in this case."

A further observation of such cases inclines me to the view that this "terminal dysentery" is, to use Sir William Gull's words, "*rather a mode of dying than a cause of death.*" Sir William Gull made use of this expression with reference to pneumonia, which in the Registrar-General's returns is responsible for a large number of deaths. Sir William Broadbent, in quoting this expression (*Practitioner*, Jan., 1900, p. 11), goes

on to say: "Pneumonia, sometimes of a very acute type, may constitute the closing scene in old age, or in debilitated constitutions, or may step in at a late stage of many acute or subacute diseases—in smallpox and the fevers generally, in acute rheumatism, in renal disease, or it may attack an apparently strong and healthy person at any period of life." That is to say, it is in many cases "terminal pneumonia." This is exactly the position I take up about "terminal dysentery." Just as people in Europe "die at their lungs," so in the tropics, and in Bengal especially, people "die at their bowels." It is so well known that bowel complaints frequently supervene at the end of many diseases in Bengal that many years ago Norman Chevers proposed the name *Morbus Bengalensis*. It is probable, therefore, that Dr. Manson's explanation is true; the germ of dysentery is everywhere present in the tropics, just as the germ of pneumonia is everywhere present in England, and finds in these dying persons a favourable soil to assert its virulence, and hasten the end, the dysentery being "rather the mode of dying than the cause of death." The fact that the symptoms are less acute—that, in fact, the dysentery is usually only discovered by an examination of the stools (a strictly enforced routine rule in all Indian jails)—is explained by the fact that acute symptoms are less marked owing to the diminished vitality of the patient, who has been suffering for weeks or months from some debilitating and cachectic disease. It is not the dysentery which kills the patient; he has obviously been dying from some other disease.

Brief notes only are given of the following five cases to illustrate the above remarks, but the exact date of the supervention of the terminal dysentery is noted in each case. In my experience it is an invariably fatal symptom; I have never seen a patient recover in which it has occurred.

CASE I.—Shk. Aklu, sent to jail in bad health, in January, 1899. Admitted to hospital for the last time, on March 25, emaciated, anæmic, persistent vomiting and inability to retain food, dilated stomach, stools soft and liquid, but without blood or mucus; remained under treatment for dilated stomach. The first appearance of dysentery was noted on April 28, he passed regular "meat-washings" stools up till his death on May 6, *i.e.*, terminal dysentery appeared nine days before death.

Post-mortem.—Great emaciation, organs healthy, stomach much dilated, with partial closure and ulceration of the pyloric end. In large intestine recent ulceration of a gangrenous type.

CASE II.—Paria, a female life prisoner, aged 65 years, many years in jail. In hospital for better care, owing to old age and general debility. The symptoms of terminal dysentery appeared twenty-one days before death.

Post-mortem.—Ulceration of great intestine, which had also ulcerated into the posterior wall of the vagina.

CASE III.—Sk. Hossaini, frequent admissions to hospital for malarial fever and bronchitis. For several weeks before death low irregular fever (probably the "secondary fever" of the malarial cachectic described by Major R. Ross, I.M.S.), heart found dilated, double

the size of the man's fist, no pneumonia, no tubercle of lungs, spleen much enlarged, hard and fibrous (malarial). In large intestine recent patches of dysentery, which supervened only fifteen days before death, when he was clearly dying from advanced malarial cachexia.

CASE IV.—Surja Gwala, aged 30, in jail four years, several previous admissions from malarial fever and anæmia. Tubercle of lung diagnosed in October, 1898. Had chronic hectic fever for one month before death, died July 7; the terminal dysentery noted on June 26, or eleven days before death.

Post-mortem.—Lungs, thick fibrinous adhesions on both sides, pericardial sac obliterated with fibrinous pericarditis; lungs, a mass of miliary tubercles; spleen much enlarged; recent fresh ulceration of large intestine.

CASE V.—Jitu Mahomed, aged 42, one year in jail, came direct to the hospital on admission to jail, in an advanced state of malarial cachexia, very anæmic, enlarged spleen, irregular fever of the usual secondary type, with irregular intervals lasting for five or six days. Had been continuously in hospital for five months when the terminal dysentery appeared, fourteen days before death.

Post-mortem.—Great emaciation, no œdema of lungs, no tubercle, no pneumonia. One large irregular patch on lower surface of liver, due to gall-bladder staining. Spleen much enlarged, weighed 4lbs., hard and fibrous. In small intestine several round worms, in large intestine the four lower feet were thickly covered with fresh diphtheritic ulceration. The symptoms of dysentery had only supervened fourteen days before death.

I have another case at present in hospital, a Bengali, sent here from a Calcutta jail for the benefit of his health. He has been suffering from tubercle of the lungs for many months, with fever and progressing emaciation, but a few days ago blood and mucus were found in his stools, and I have no doubt this means terminal dysentery and that he has a very short time to live.

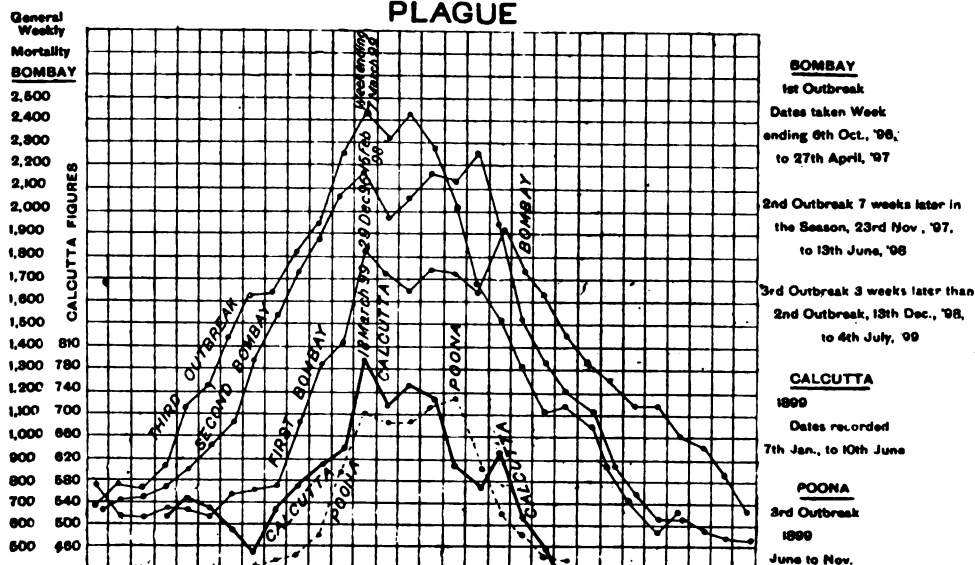
OBSERVATIONS ON THE EPIDEMIOLOGY OF PLAGUE.

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THE accompanying chart shows the courses of the three successive outbreaks of plague in Bombay city and of one in Poona, and for comparison, but on a different scale, the course of the second outbreak of plague in Calcutta. The returns of the weekly general

mortality are taken because these seem the most reliable estimate of the extent of the epidemic; the excess mortality above the average being for all practical purposes the amount of the plague. The official returns of plague are so utterly untrustworthy that in the absence of any other cause for an increased death-rate the excess mortality above the normal becomes the only guide. With regard to Bombay it will be seen that each outbreak occupied about the same period of time. There is in each case the same rapid rise, the same fluctuating summit of highest mortality, and the same correspondingly rapid fall. The charts of each outbreak have been super-imposed in order to emphasise the corresponding weeks of highest mortality, but it must be clearly understood that these highest points did not occur at the same dates in successive years. The second outbreak in

PLAGUE



Bombay was seven weeks later in the season than the first, and the third occurred about three weeks later in the season than the second. Although the first outbreak culminated at the coolest period of the year, the second rose to its highest point as the weather was getting warmer, and the third outbreak showed its still higher range of mortality in the hot month of March. Again the outbreaks in Calcutta and Karachi occurred during the hottest months of the year, that at Poona during the monsoon, and the Southern Mahratta country was devastated at the close of the monsoon and while the winter was coming on. These facts show that season and temperature have little or no influence in determining the rise or fall of an outbreak.

It will be observed that there is a break in the steady and continuous rise of mortality in the first and third outbreaks in Bombay. There is also a break during the fall of the same two outbreaks. In the Calcutta curve there is also a break in the rise, and a very marked secondary rise for one week during the months of the fall. It is suggested that these are not accidental, but there is nothing upon which to ground an explanation. The fluctuating summit of highest mortality in each outbreak lasting

from four to six weeks is very peculiar. The severity of each outbreak in Bombay can be roughly estimated, as the average weekly mortality is between five and six hundred, and on this basis it is assumed that about 22,000 lives have been lost each year from plague alone. These characteristics of a plague outbreak in a large city are reproduced in the chart prepared from the mortality returns of the city of Poona, and to a certain degree also in that for the city of Karachi. The mortality in Bombay city at each outbreak, in Karachi, and in the previous outbreak in Poona, has been roughly estimated at one in forty of the population, but in the last and third outbreak in Poona it has been much more severe—more than equalling in its devastating effects the two previous attacks combined—and carrying off nearly one in every ten of the estimated population. There appears to be no explanation to account for this, and there is no parallel to it in any part of India. The chart for Poona, drawn on the same scale as that for Bombay, is very similar. Considering that this city has only about a sixth of the population of Bombay, the terrible severity of the last outbreak is very evident. In comparing all the lines it will be seen that the week of highest mortality occurred in three of the outbreaks immediately before the decline.

CALCUTTA	POONA	BOMBAY		
		1st outbreak	2nd outbreak	3rd outbreak
Week ending Jan. 7, '99, and following weeks	Week ending June 16, '99, and following weeks	Week ending Sept. 1, '96, and following weeks	Week ending Oct. 19, '97, and following weeks	Week ending Nov. 8, '98, and following weeks
562	126	667	776	645
520	159	593	754	604
543	205	618	741	606
521	302	647	701	616
483	357	720	700	679
450	548	791	682	688
532	815	634	704	798
572	1102	606	706	792
604	1082	698	785	894
628	1087	668	835	1111
799	1112	623	975	1239
716	1182	704	1061	1448
743	889	760	1307	1601
722	635	772	1540	1612
619	554	1051	1726	1808
562	420	1310	1871	1907
631	305	1416	2067	2249
517	241	1853	2195	2431
443	125	1711	1974	2312
387	109	1639	2080	2408
436	76	1758	2184	2290
435	..	1726	2197	2010
372	..	1645	2268	1696
..	..	1911	1938	1503
..	..	1728	1519	1305
..	..	1640	1303	1099
..	..	1465	1202	1127
..	..	1326	1116	1047
..	..	1258	872	890
..	..	1139	725	711
..	..	1141	616	594
..	..	1007	633*	609*
..	..	970	589	..
..	..	836	525	..
..	..	671	504	..
..	..	638	448	..
..	..	558	500	..

* Doubtful.

Another point of interest, but which is not shown by the chart, is the heightened general mortality for weeks and months before the outbreak definitely shows itself. The decline, moreover, of each attack, though rapid at first, terminates slowly, and a plague outbreak seems to linger for many weeks or even months before finally disappearing. In Bombay city, however, it is doubtful whether there has been a single week clear since the declaration of plague in September, 1896.

With reference to the various diseases making up the general mortality, it is important to note that other epidemic diseases were conspicuous by their absence. Cholera, dysentery, epidemic diarrhoea, and fevers generally were all, as far as imperfect registration indicates, considerably below the average. In Bombay, relapsing fever was the only concurrent fever and in each of the above towns the mortality from cholera was exceptionally low.

It only remains to give the figures of the weekly general mortality upon which the accompanying chart is based. (See table).

NOTES ON BERI-BERI IN THE AUSTRALIAN PEARLING FLEET, 1883 TO 1887.

By T. H. HAYNES, Esq.

Ship.	Hands	Duration of Voyage in Months	Deaths	Cases	Diet	Date of First Death
<i>Naked Diving.</i>						
1. Flowerdale.	80	7	19	Very many	Rice and fish.	—
2. Sree Pas Sair.	7	10	None	None ..	—	—
3. Ditto ..	70	5	16	Very many	Ditto ..	4th month
	77	8	7	30	Ditto; changed in fifth month to mixed diet.	3rd month
<i>Dress Diving.</i>						
4. Sree Pas Sair.	70	7	None	None ..	Mixed diet.	—
5. Ditto ..	84	7	None	Symptoms in the last month.	Ditto ..	—
6. Ditto ..	118	9	3	Ten. Taken home in eighth month.	Ditto ..	7th month

These notes refer to certain Malay crews, the first lot of which were employed in naked diving, and the second lot as pumpers for apparatus diving. All of them were shipped at Koepang, in the Dutch Island of Timor, with the exception of the seven men noted separately in No. 2 and the seventy-seven in No. 3, all of whom came from Sooloo, in the Philippine Archipelago, and were of finer race. The immunity of many previous crews from beri-beri, and the prevalence of the disease amongst the Malay crews in the patrol vessels of the Netherlands Indian Government, supports the opinion which obtains, that the great difference between the severe and constant work of diving, and the comparatively easy work of pumping with five or six days idleness every spring-tide, was not a factor of any importance; moreover, in the

numerous cases of Australian natives employed in naked diving and fed upon "damper," the men were entirely exempt from the disease.

The conditions of ventilation at night differed, but in manner favourable rather to the Malay divers, who were employed only during the hot months and slept with open hatches; whereas in the case of the Australians the hatches were at night generally kept nearly all on, and in the case of the Malay pumpers working at any season of the year, they slept in very confined quarters on board small luggers of 5 to 10 tons.

The apparatus-boats also carried a number of Malays, Japanese, and South Sea Islanders as divers, who were shipped separately at irregular intervals; but as these men not only enjoyed much better rations, but also obtained considerable quantities of beer and spirits, they are purposely omitted from the tabulated figures. As a rule, however, they were exempt from beri-beri, and the South Sea men, who are bred on yams, appeared entirely proof against it, as also were the Europeans.

Two forms of the disease present themselves—the ordinary form, exhibiting dropsical swellings of the arms, legs, and abdomen, short breath, weak pulse, palpitation of the heart, pallid appearance, stiffness of the legs, and constipation. The stools are of the colour of teak-wood and this sign is regarded as a precursor of the other indications of the disease. Ordinary cases of this nature exhibit no particular change for days and weeks, and frequently recover.

The second form is extremely rapid. A man will come complaining say, at eight o'clock in the morning; ashen appearance, pallid lips and gums, yellowish conjunctivæ, an imperceptible pulse, palpitation, laboured and painful respiration, with pain indicated as central below the breast-bone and over the heart. In such cases death usually ensues by 4 p.m. the same day.

Regarding the tabulated figures in simultaneous voyages, marked No. 1 and 2, the outbreaks on board the *Flowerdale* and *Sree Pas Sair* were very severe, and partook distinctly of the rapid form, particularly at first. The work on board the *Flowerdale* was constant and heavy, but on the other ship it was much lighter than usual. None of the seven Sooloo men, however, who had shipped two months earlier, showed any sign of the complaint, although they were not sent home until three months after the others.

During voyage No. 3, lasting from September, 1884, until May, 1885, the first death occurred on December 15. This was Ipajaty, the only man of poor physique on board. He had been a half-starved slave, and as he was unable to dive at all his work was very light, and as probably for the first time in his life he had unlimited quantities of rice and fish, he increased remarkably in weight. The disease seized him in its rapid form, and he died within a few hours. The ship's company included three of the old hands from the previous voyage, but no further cases appeared until February, when a large number of cases of the ordinary kind developed. The three meals of rice were then reduced to one, and in addition to fresh fish a mixed diet of flour, beans, peas, with a few potatoes and onions and plenty of turtle eggs, was substituted, and all cases were put into a camp ashore, and walking exercise enforced. Aperients were ad-

ministered plentifully, and gin and quinine served out. In February two deaths occurred, in one of which there was an inability to pass water, and on March 15 another death took place, and as some thirty cases were in camp it was decided to abandon the cruise and send the men home. In all other respects there was no illness beyond one case of shingles, two of fever, one of severe piles, at first mistaken for dysentery, but quickly cured by balsam of copaiba—and one inscrutable case of emaciation—a man called Adam, who was said to be troubled with a worm. The announcement of returning home so raised the spirits of the men in camp, that on reaching the port of Cossack three days later, preparatory to clearing for the Philippines, the improvement was so extraordinary that it seemed ridiculous to abandon the cruise. During the week's stay in Cossack fresh mutton was supplied in abundance, and on March 25 the ship cleared out with no one on the sick list at all. Congenial occupation was afforded by tending a deck load of live stock, and all went well until April 1, when Adam complained of feeling ill in the morning, and died in the afternoon; the next day another man followed suit, and on the third yet another, after which there was no further illness.

During cruise No. 4 a mixed diet was adopted, and thirty sheep turned out on an island afforded an occasional taste of fresh meat. It had by this time been agreed that every endeavour should be made to return each batch of men within seven months, and on acting on this stipulation during the No. 4 cruise, the result was—no appearance of beri-beri at all.

The fifth batch were also returned home safely in the seventh month, although the bulk of the rice used during the earlier period of the cruise had suffered by sea-water. During the last month two men showed symptoms of the disease, but reached home safely, aided by a liberal supply of mutton, potatoes and beer.

The sixth cruise lasted nine months, from November, 1886, to August, 1887. In April the stores were again damaged by sea-water, but eventually replaced. On the morning of June 9, the seventh month, a man was taken with the disease, showing all the symptoms of its rapid form, and slight swelling of one leg; he died at 7 p.m. On June 27 the death occurred of a boy who had become extremely emaciated after several days of high fever, parched mouth and extreme thirst: it is doubtful whether this was a case of beri-beri at all. A schooner was then sent to Koe-pang for fresh men, and it was decided to return ten men who showed dropsical symptoms; of these one died suddenly on July 3, while watching a game of cards in the hold, and the other nine reached home safely. The remainder, less seventeen drowned, were returned in August with no signs of beri-beri at all.

Nothing has been reported from subsequent voyages to alter the opinion—

(1) That beri-beri is confined to a very great extent to rice-eating races, and with proper care will not develop in less than seven months.

(2) That the substitution of a mixed diet of wheat-flour, beans, potatoes, &c., to the exclusion of rice, mitigates, even if it does not prevent, the disease.

Lime juice does not appear to be of much service,

but beer is most beneficial. Finally, the recovery of any man afflicted with beri-beri, who survives twenty-four hours after return home, where he obtains fruit and resumes his former mode of life, may be regarded as certain, and a few months' residence in his native place will render him eligible for a further period of service.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By GEORGE H. F. NUTTALL, M.D., Ph.D.
Pathological Laboratory, Cambridge.

THE view that mosquitoes¹ serve as vehicles for the malarial infection has long been entertained in various parts of the world. Barker (1900, Feb. 3, p. 238), writing of malaria in the Philippines, says, "Not uninteresting, too, in passing, is the statement in certain Jesuitical records of Mindanao, that the natives of that island recognised as far back as two centuries ago a relation between the intermittent fevers and the prevalence of mosquitoes." Sforza (1899) as also Grassi (1899, August 31, p. 12) state that Lancisi (1717) considered mosquitoes served to propagate malarial infection. I have not as yet been able to verify this interesting reference. (See bibliography at the end of this publication.) When visiting Florence in February, 1899, Professor Lustig informed me that the mosquito-hypothesis had long prevailed amongst the Italian peasants, and Geheimrath Rubner also tells me that the same belief exists in Southern Tyrol. Koch (1898, i.), in a report on his observations in German East Africa, states that the negro of the Usambara Mountains, who acquires malaria when he descends to the lowlands, has also convictions on the subject. "He calls the disease Mbu, and if one asks him where he has acquired it, he replies that there are insects down there which are also called Mbu (*i.e.*, mosquitoes) like the disease—these had stung him, and that is how he had acquired the disease." Dr. Ronald Ross wrote to me, in a letter dated October 31, 1898, that a Mr. Jameson in Assam, who has also been in Africa, has informed him that in parts of Africa and Assam the natives believe that mosquito-bites cause fever. The mosquito-malaria theory has certainly existed a long time in the United States.² It has long been known, and this in different parts of the world, that curtains, gauze veils, mosquito-nets and the like, protect against malarial infection.

¹ By the term mosquito is meant a variety of different insects—see below.

² In King's publication (1883) there occurs a reference to a paper by John Crawford, "Mosquitoal Origin of Malarial Disease," which had remained inaccessible to King, and was said to have appeared in the *Baltimore Observer*, in 1807. Nicolas (1889) also gives this reference which he must have taken from King. Bignami (1896) seems to have taken it from Nicolas; the latter gives the wrong date, *i.e.*, 1867 instead of 1807. It seemed to me a matter of considerable interest to follow up this reference, so I wrote to Professor King about it. He has been to a great deal of trouble and has found out that the *Baltimore Observer*, which only appeared in 1806-1807, contains no such article. That Dr. John Crawford considered that insects played a rôle in the spread of disease, has been noted

In 1848³ Nott, of New Orleans, published an essay on yellow fever in which he also refers to malaria as if the mosquito theory had already been advanced, and he gives grounds for his belief that the mosquito also gives rise to yellow fever. In 1883 a most elaborately stated argument was published by King, in which he brings together a mass of evidence on the subject, vastly more, in fact, than other authors have since gathered, and I shall often have occasion to refer to his paper. It is curious to look over the more recent literature on the subject to see how writers have rediscovered the mosquito-malarial theory. In France the theory is ascribed to Laveran, in Germany to Koch and Pfeiffer, in England to Manson, whilst in Italy the names of Bignami, Mendini and lastly, Grassi, are identified with it. By far the most masterly exposition of the theory was written by King. It is first mentioned by Laveran in 1891, by Manson in 1894, whilst Pfeiffer makes the first published statement of Koch's views in 1892. As far as I can gather, Bignami and Mendini refer to it in 1896 and Grassi in 1898. It is difficult to say to whom is really due the most credit. I shall not attempt to do so, but will leave it to the reader to draw his own conclusions from the facts here presented.

Laveran, in 1891, expressed his belief that malaria was conveyed by mosquitoes, and gave some of the usual arguments in support of the theory. In the same year Flügge⁴ wrote "Manche Beobachtungen, so z. B. die Erfahrung, dass die Abendund Nachtluft vorzugsweise Gefahr bringt, während über Tag die Luft desselben Ortes gar nicht oder selten Infektionen veranlasst, ferner dass oft nach flüchtigsten Aufenthalt auf Malariaterrain sehr rasch Infektion eintritt, legen die Vermuthung nahe, dass der Transport der Erreger zum Theil durch Insekten, namentlich Mücken, Mosquitos, &c., besorgt wird. Diese sind einer solchen Rolle offenbar sehr geeignet, schwärmen vorzugsweise Abends und Nachts und sind eventuell im stande, die Erreger direkt ins Blut einzupfropfen, und so eine Erklärung für die Fälle zu liefern, in welchen schon wenige Stunden nach der Ankunft auf dem Malariaterrain Erkrankung eintritt." Bignami, as also Mendini, writing in 1896, express similar views.

Prof. Robert Koch has had the kindness to inform me that the possibility of mosquitoes playing a rôle in malaria first occurred to him whilst he was in India in 1883 to 1884, when he had an opportunity of studying the conditions under which tropical malaria occurs. Since then he has always referred to it in his lectures. The first reference, in the literature of his views, is by Pfeiffer (1892), who writes "Es wäre

under the heading "Miscellaneous." Both Prof. W. S. Thayer and Prof. A. F. A. King looked through a review of Crawford's papers which appeared in the *Baltimore Medical and Phys. Recorder*, 1809, but they found no mention therein of mosquitoes in connection with malaria. I am exceedingly indebted to both the gentlemen above named for the readiness with which they assisted me.

³ I take this opportunity of thanking Dr. Isadore Dyer, of New Orleans, La., for the trouble he took to write out and send me an abstract of Nott's publication, the original being inaccessible to me.

⁴ Flügge, C. (1891) *Grundriss der Hygiene*, pp. 473 and 532.

möglich, dass auch bei den Malariaparasiten exogene Zustände existiren, Entwicklungszyklen, die ausserhalb des menschlichen Körpers, vielleicht im Leibe niederer Thiere (gewisser Insekten z. B.) vielleicht auch zum Theil mindestens im Boden sich abspielten. Diese exogenen Malariakeimen können dann durch die Luft, durch das Wasser oder, worauf Robert Koch mich aufmerksam machte, durch den Stich blutsaugender Insekten auf den Menschen übertragen werden." This is the first mention of this subject by Pfeiffer, and Manson first speaks of the probable relation of mosquitoes to malaria in a publication which appeared in 1894. He drew an analogy (as King had done) between the role of the mosquito in relation to *Filaria sanguinis hominis* and its possible role in malaria, where he considered that the flagellated form of the malaria parasite represented the first phase in the extracorporeal existence of the latter. These views of Manson's will be again considered below.

King¹ (1883) very properly introduced the arguments which he had gathered in favour of the Mosquito-Malaria Theory with the words, "While the data to be presented cannot be held to prove the theory, they may go so far as to initiate and encourage experiments and observations by which the truth or fallacy of the views held may be demonstrated." I can do no better than quote his words. In the following pages I have incorporated most of King's arguments, and added many data gathered from other writers, as well as some suggestions of my own.

EVIDENCE IN FAVOUR OF THE MOSQUITO-MALARIA THEORY.

1. *Malarial Season*.—The malarial season corresponds usually to a season of *warmth* and *moisture*, conditions which are most favorable for the development of the mosquito. Malaria is rarely developed at a temperature below 15 to 16° C., a temperature which is necessary for the evolution of the mosquito, and it is checked at 0° C., at which temperature the mosquito is inactive. (Hirsch,² King, &c.) In many places malaria develops after the first rains; the latter may have formed pools in which mosquitoes multiply. Malaria disappears when the rains subside (Bignami) and so do mosquitoes. Malaria often ceases after excessive rains (Hirsch), for then the pools are often washed out and flooded; besides that, excessive rains are always injurious to insect life.³ Cooke (*Tran-*

sylvanian Journal of Med., 1828, i, 341, cited by Hirsch, p. 182) wrote: "Wet summers are sickly and dry summers are healthy." . . . except in the neighbourhood of marshes, ponds and rivers." The prevalence of malaria in wet years has been observed in many places. (See further, Hirsch, p. 182. This author, by the way, makes no mention of the mosquito-malaria theory. [See also, Laveran (1878), p. 28]). In rainy years there would be a better opportunity for the multiplication of mosquitoes, more pools being formed.

2. *Malarial Country*.—Low, moist places, swamps, jungles, the low seaboard and river estuaries and valleys, especially after inundations have subsided (the Nile, Indus, Euphrates, Ganges and Mississippi valleys, &c.), are the chief localities affected. (Hirsch, Laveran, 1898, &c.). Mosquitoes abound in such places, and require pools or almost stagnant bodies of water in which to multiply. Malaria is most abundant as we approach the equator, where insects are also most numerous throughout the year.⁴ In countries where irrigation has been introduced without regard to efficient drainage, an outbreak of malaria, or an increase in the severity of the cases, has followed. We have an example in Southern California. (Welch and Thayer, 1897, p. 97. Hirsch also refers to such cases).

3. *Conditions which afford protection against malaria and—mosquitoes*.

Protection of the body. That closing the windows and doors at night, as well as the use of mosquito nets, gauze veils, curtains, &c., protect against malaria is a matter of long experience in malarious countries. Johnson (1818), Macculloch (1827), Brocchi cited by Goode (1834) and Evans (1837) refer to the protection afforded by the use of gauze or fine cloth at night. Macculloch writes that by surrounding the head with a gauze veil or canopeum, the action of malaria is prevented, and that it is even possible to sleep in the most pernicious parts of Italy without hazard of fever. Day advises the use of mosquito curtains "through which malaria can seldom or never pass." Oldham (1871) states that the Jeevas of the Punjab, who are employed in fishing and catching wild fowl, spend the whole night in their boats, under the reeds of the marshes, "unharmful in the midst of malaria:" but they are wrapped from "head to foot" in a peculiar costume that completely envelops them, and which they always put on at sunset; and, moreover, a smouldering fire is kept up in the boat. (Quoted from King). Bignami (1896) states, as we all know, that the inhabitants of malarial districts avoid going out at night or sleeping in the open air. They close their windows and doors tight and use mosquito bars. He writes that it is known that covering the skin is a protection, and relates the case of a Russian physician, who had never acquired malaria in malarial countries, because he always slept with gloves and a mask. He states that Emin Pasha always took mosquito nets with him on his African journeys, considering that

¹ King thinks mosquitoes may even be protected against malaria by their inoculating an attenuated virus. Koch (1898) expresses the same idea. It is worthy of note in this connection that Koch observed a mild form of Texas-fever in cattle on which had been placed young ticks which had been exposed to unfavorable conditions.

² Hirsch, Handb. d. hist.-geogr. Pathologie, vol. i., Stuttgart, 1881.

³ It seems almost superfluous to cite authority for this statement, the observation having been so frequently made. Von Nordenskiöld ("Grönland, Schilderung der zweiten Dickson'schen Expedition ausgeführt im Jahre 1883." Leipzig, 1886, p. 75 and 236), who tells of the sufferings due to the enormous quantities of *Culex* on the coast of Greenland, observed that continued rains greatly reduced their numbers. They were also not

troublesome whilst a fresh sea-breeze was blowing. Weeks (1890) states that heavy showers frequently destroy great numbers of dragonflies. I might add that rain and wind do not only affect the winged mosquito, they will also prevent many from escaping from the puparium, and the process of egg-laying.

⁴ The fact that malaria does not occur in northern countries, where mosquitoes are also at times found in great numbers, may be due to the low temperature there, hindering the development of the malarial parasite in the body of the insect. Besides that, the northern species of mosquitoes may be unsuitable hosts,

they kept off malaria, and he cites Nicolas ("Hygiene of Camps and Marshy Places") as writing, "Without attributing to the puncture of mosquitoes any relation whatever with the microbes of the fever, one may be certain that irritation by them produces sleeplessness and predisposes to fever." Quite recently Koch (i. and iii., 1898) has come forward in favour of the mosquito-malaria theory, and advises the use of mosquito-nets, screens, curtains, and clothing, which is impenetrable to the proboscis of the mosquito, as well as quinine as a prophylactic measure. King (1883) recommended all of these.

Agglomerations of Houses exclude Malaria.—Malaria does not penetrate into cities situated in malarial districts, because the mosquitoes are stopped by walls, hedges, &c., and are attracted by the lights in the suburbs. The mosquito-malaria theory offers an explanation as to why people living on one side of the road are attacked by malaria, while those on the other side escape, "as on the high road between Chatham and Faversham" (Macculloch). The same thing has been observed in Civit  Vecchia (Johnson, references cited from King.) Jilek ("Ueber die Ursache der Malaria in Pola," Wien, 1868, referred to by Hirsch) showed that especially those parts of the town of Pola which were most exposed to the winds from the neighbouring swamps were affected by malaria. Wilcocks (*American Journ. of the Med. Sc.*, January, 1847, Hirsch) observed in the severe malaria epidemic which visited Philadelphia in 1846, that the disease affected almost exclusively persons who lived in streets or rows of houses exposed to the south wind. Laveran (1896) cites Mendini as saying that the central parts of Rome are healthy because they are free from mosquitoes. During the malarial season only those persons who venture out of the city walls acquire malaria (Laveran (1898), p. 9).

Protection attributed to intervening Woods or Expanses of Water.—It has been claimed that woods have the power of obstructing or preventing the transmission of malaria by the wind. In other words, they have been said to hold back the mosquitoes that are blown or fly there from a malarial foyer. This has apparently been best demonstrated by the influences denudation and planting have had upon the health of communities. Coons (*Transylvanian Journal of Medicine*, ii., p. 112, Hirsch, p. 208) reports the following regarding the malaria epidemic of 1826 in Alabama: In the vicinity of Moulton, and situated half a mile from a swampy lake, was a large farm on which all the people had previously been healthy. A thick wood, which lay between the lake and the farm, was cut down. This wood had hitherto served as a barrier to the winds coming from the lake. Of 150 persons living on the farm only 3 or 4 escaped the malarial infection. Wooten (Lewis, "Medical History of Alabama," p. 17) reports a similar case, that of a plantation which was separated by a dense wood from a creek which flowed between swampy banks, and was situated about a quarter of a mile away. In the winter of 1842 to 1843 the wood was cut down, with the result that already in the following summer the negroes on the plantation who had previously been healthy, were severely visited by malaria. The owner of the plantation was compelled to transfer the negroes

to the other side of the creek, the bank on that side being separated from the new settlement by a wood. The result was that the cases of malaria decreased and people living on that site remained healthy. Sir Francis Day (cited by King) wrote: "Malaria may be carried by the winds to places where it was not generated; it is obstructed by and hangs in the foliage of trees, or in mosquito curtains; it subsides into low places, and may be blown over a hill, and may be very virulent on the side opposite to that on which it was formed. In like manner it may be taken up the side of a hill, and, as a lull takes place in the atmosphere, consequent upon its weight, it rolls down and may thus envelop its base with a deadly belt of fever, for there, hanging in the leaves of the trees, it gradually sinks through them to the earth beneath, in which situation it is most dangerous to pass the night." Mondineau (*La pathologie et l'hygi ne des Landes*, Paris, 1867, Hirsch, p. 209) writes: "One thing is certain, that in the wastes of the canton of Houielles intermittent fevers have become much rarer, and especially much milder, since great forests of pines have come to form a natural barrier to the propagation of the miasma." Dods (1878) says he has never noticed that persons living in the vicinity of rank vegetation suffer particularly from malaria as long as the growth is left undisturbed. He notes the unhealthiness of the Assam tea-gardens when the virgin forest is first cleared away. It is the clearings in Borneo forests that are unsafe, protection being afforded by keeping in the forest to the windward of the clearings. He advises when the soil is turned to scatter lime¹ on it, or if possible to beat it down and cover it with turf. It is needless to multiply the references to the literature on the subject, or to dwell on the fact that the planting of trees also modifies the drainage and moisture of the soil.

(To be continued.)

THE ENDEMIC CENTRES OF PLAGUE.

By FRANK G. CLEMOW, M.D., D.P.H.

I.

PLAGUE IN SOUTHERN CHINA.

I HAVE selected for the title of these papers the words "The Endemic Centres of Plague." Some explanation of the limited sense in which the term "endemic centre" will be here employed is, however, necessary. Since the year 1894 plague has spread to Canton, Hong-Kong, Formosa, India, Mauritius, Madagascar, Egypt, Portugal, South America, Australasia and elsewhere, and in many of these places the disease has already become endemic. But with these areas of its prevalence I do not propose here to deal, and these papers will be confined to a discussion of those geographical centres where plague has been truly endemic, or has recurred from time to time, during the latter half of the present (nineteenth) century, but prior to the year 1894.

The material for this discussion was collected in the course of an inquiry directed to ascertain what relation the wide diffusion of plague since the year

¹ See more about the effect of lime further on.

1894 has borne to the centres of the disease which existed before that date. It can scarcely admit of doubt that the year named was a real epoch in the history of plague. For many decades before the disease had been confined within comparatively narrow limits. In China, in the Himalayas, in Mesopotamia, in Persia, in Arabia and in Tripoli, centres of plague were known to exist, and had always been regarded as possible sources of danger in the future; and recent inquiry has further brought to light the fact that similar foci of the disease have, at least for some years past, been more or less active in Siberia, in Mongolia and in Central Africa. In each of these centres the area of disease prevalence was comparatively small, and the infection rarely, or in most cases never, showed any tendency to transcend those limits. But in 1894 there was a sudden and marked change in the behaviour of the disease. It broke out violently in parts of China hitherto quite free from it, and its subsequent spread to the other countries named above has shown that the infection had acquired an immense power of diffusion—a power which for a very long period it seemed to have lost. At the present moment plague is more widely diffused over the earth's surface than it has ever before been in historical times. The statement sounds alarming, but it is nevertheless literally true. Fortunately, the disease, though very widely diffused, has, in the majority of countries outside China to which it has been imported, shown no tendency to spread widely in those countries, and, with the exception of India, it has nowhere caused an epidemic of extreme intensity or a mortality in any way comparable to the appalling mortality of the Black Death of the fourteenth century.

In discussing the endemic centres of plague existing before 1894, and the history of the disease since that year, the questions of greatest importance are the following:—Can the recent spread of plague be traced to one or some of those pre-existing centres? What has been the behaviour of the disease in those centres since 1894 as compared with its behaviour before that year? Has there been any synchronous revival in activity of the disease in these centres during recent years, indicating some universal or earth change affecting widely distant centres at or near the same time? or, has there, on the other hand, been a spread of the disease from one centre only, upon which its subsequent wide diffusion has been directly or indirectly dependent? A careful study of the various centres in question goes to show that the last-named supposition is the correct one; that there has been no synchronous revival of activity in the scattered parts of the world where plague existed before 1894; and that its spread since can be traced with almost certainty to a direct or indirect diffusion of infection from the important endemic centre or centres of plague in Southern China.

Plague has for a long period been endemic in Yunnan, the province in the extreme south-west corner of the Chinese empire, closely bordering on Burmah on the west, and extending northwards towards the frontiers of Tibet. It was until recently believed that the existence of plague in this province could be traced back with certainty only for some half century or less, but a reference to a Chinese writer has recently been

brought forward which scarcely leaves room for doubt that it has existed there for a much longer period.

A Chinese gentleman has quoted in the pages of *Nature*,¹ a passage in a Chinese work which he found in the library of the British Museum; and which, he says "bears witness to the much earlier occurrence of the pest in Yunnan [than is currently supposed,] insomuch as the author, who was born in 1736, and died in 1809, speaks of his contemporary, dead thereby [thus]:—'Shi-Tau-Nan, the son of Shi-Fan, now the governor of Wang-Kiang, was notorious for his (poetic) gift, and was only thirty-six years old when he died. . . . Then in Chau Chau [in Yunnan] it happened that in the daytime strange rats appeared in the houses, and lying down on the ground perished with blood-spitting. There was not a man who escaped the instantaneous death after being infected with the miasma. Tau-Nan composed thereon a poem entitled "Death of Rats"—the master-piece of his; and a few days after he himself died of this queer rat epidemic.'" The close association of this peculiar behaviour of rats with the prevalence of plague in man, observed in the same province in recent years, makes it almost certain that the disease here referred to was plague, although no symptoms are mentioned by which it can be finally identified. Accepting this view, it will be seen that so long ago as the latter half of the last century (or at latest within the first decade of the present century) plague was prevalent in Yunnan. It may even have existed earlier, for a French missionary has stated that he read "in the itineraries of Jesuits sent by the Emperor K'ang-Hsi to survey the province [of Yunnan] in 1617, the description of a disease quite similar to the *yang-tzu-ping* [plague] observed nowadays."

Coming to more recent times, the first positive reference to plague in China relates to the year 1844; in that year plague is said to have been epidemic there for the first time in contemporary history.²

The most authoritative accounts of this centre of plague are those of Rocher,³ of Baber,⁴ and more recently of Michoud.⁵ From these writers it may be gathered that the disease has been most active since the time of the great Muhammadan rebellion in the late fifties and early sixties. In regard to its source little is known with certainty. Rocher says

¹ *Nature*, February 16, 1899, Mr. Kumagusu Minakata. The name of the Chinese work from which the passage is quoted is given as "Peh-Kiang-Shi Hwa," and that of the author as Hung-Liang-Ki.

² "Medical Reports of the Imperial Maritime Customs of China," Second Series, 48th issue, Shanghai, 1895. The statement of the missionary was made to Dr. Michoud, and is quoted in his "Report on the Health of Mengtze" in that volume.

³ Lowson, *Indian Medical Gazette*, January, 1897. The authority for the date is not quoted. In regard to the history of previous plague prevalence in China, Dr. Lowson asked Mr. J. Dyer Ball to examine the Chinese literature of the subject, but after months of diligent inquiry, that gentleman was unable to trace any record of an epidemic sufficiently severe to leave a mark upon history.

⁴ "La Province Chinoise de Yunnan," par Emile Rocher, de l'Administration des Douanes Impériales de Chine, Paris, 1879. "Notes sur la peste au Yunnan," form an appendix to the second volume of this work.

⁵ Parliamentary Papers, China, No. 3 (1878), pp. 22, 23, quoted from a report by Mr. Baber upon the route followed by a certain mission between Tali-fu and Momein.

⁶ *Loc. cit.*

that the Mandarins believed it came from Burmah by caravans. Proof of its prevalence in Burmah, however, appears to be wanting. Michoud expresses the belief that it may have come through Tibet from India, and may be, in fact, the *Mahamari* itself, transported by caravan from Kumaon and Garhwal. On this point he writes as follows:—

"The Kumaon district is contiguous to Western Tibet, and though the distance which separates Yunnan from Kumaon is not less than 1,500 miles, strong reasons can be adduced in favour of the Kumaon plague having been propagated to Yunnan through Tibet. Notwithstanding their great uncertainty, the fragments of information gathered from natives are found to agree on two points, namely, the approximate date of the appearance of the disease in Yunnan, which they represent as contemporaneous with the beginning of the Muhammadan revolt in 1860; and the fact of its having come from the west of the province. If, then, we compare the date of the first appearance of the plague in Yunnan with that of the outbreak of *Mahamari* in 1859, at the same time bearing in mind the considerable trade carried on between Western Yunnan and the anterior of Tibet by numerous Tibetan caravans coming over to take part in the great annual fair of Tali-fu, the capital of Western Yunnan, the former hot-bed of a Muhammadan rebellion, we may conclude, without being taxed with precipitation, that very likely the Yunnanese *Yang-tzu-ping* is nothing else but the Indian *Mahamari*, propagated by means of caravans all along the highlands of the southern frontier of Tibet up to the western borders of Chinese Yunnan. Eminent epidemiologists have already compared these diseases, and have observed this noteworthy peculiarity common to both, namely, the great irritability prevailing among some animals on the eve of each outbreak."

For the present this must remain a matter of surmise. No proof has as yet been adduced of this supposed origin of the Yunnan plague, and, as I pointed out in the last issue of this journal, evidence that the disease exists in Tibet appears to be wanting.

The area of prevalence of the disease in Yunnan appears to be approximately between the 100th and 104th parallels of east longitude, and the 23rd and 28th of north latitude; it is therefore very nearly, but not quite, within the tropics. A glance at a map will show that Yunnan is almost entirely made up of mountains and valleys, separated by large and approximately parallel rivers. The most important of these are the Salwen (or Lu-kiang), the Mekong, and the Kin-Shu-Kiang (the upper waters of the immense Yang-tze-Kiang). The two first-named rivers run in a southerly or south-easterly direction, and in their lower course separate, enclosing the country of Siam between them, the first flowing into the Indian Ocean, the second into the China Sea. It was in valleys lying on certain branches of these rivers that Baber described the disease as prevailing in and before 1878. He makes no mention of Mengtsz, which, according to recent writers is the principal centre of the pest. This town is situated in longitude 103° 36' east, and in latitude 23° 34' north; it lies in a plain 4,500 feet above sea level, and is

surrounded by mountains from 6,000 to 9,000 feet in height. That this has been for a very considerable time the main seat of the disease there can be little doubt.¹ Whether the area of prevalence has changed in recent years is not quite clear, but it would appear possible that such has been the case. Rocher's map of the plague area in 1871, 1872, and 1873 places it somewhat to the north and west of Mengtsz, and he makes no mention of that town. He speaks of the disease as occurring in the north and centre of the province, and it would seem that both then and now the limits of its prevalence are somewhat vague and indefinite.

The disease appears to become epidemic every year, sometimes more, sometimes less severely. It always prevails in the summer season, which is the season of the rains in Yunnan. Rocher stated that it began at the time of the rice-planting, that is to say in May or June, spread rapidly for awhile, then became less active, but gained a fresh intensity at harvest time, and from that time to the end of the year caused the greatest mortality. Michoud, on the other hand, makes no mention of this autumn recrudescence; on the contrary, he says that from September the disease disappears.

"We said," he writes, "that the epidemic appeared contemporaneously with the first rains and heat in May. As a rule, it does not last more than three or four months. In May a few cases occur. In June the disease reaches its height; 20 to 30 deaths are daily recorded. In July the epidemic begins to subside, its daily victims numbering only from 10 to 20. In August the decrease continues. In September scattered cases are met with, the latest running a mild course and being easily rescued. From September the disappearance of *Yang-tzu-ping* is complete, and until May of the next year the disease is not heard of except in extremely rare instances."

These discrepancies in statement can only be reconciled by supposing that the disease has altered its habits in the score or so of years which separate the dates at which the two authors quoted describe its behaviour. Plausibility is lent to this explanation by the fact that recent epidemics have been much less severe than the earlier ones. In former years the mortality was excessively high. Mgr. Fénouil, the Bishop of Yunnan, and a life-long resident in the country, has stated that in some years from 1,200 to 1,500 coffins passed through the gates of the city daily, and that in an average year, from 600 to 800 persons died of the disease each day. In 1866 it was believed that in and near Yunnan-fu not more than one-fifth of the former population was left alive.² In recent years, as stated above, the daily deaths have not, at the worst, exceeded 20 or 30.

All the observers quoted are agreed in stating that the disease attacks rats in large numbers, and usually before it affects human beings; and also that some of the larger animals are attacked by it. Both Rocher

¹ Michoud. See also a passage in "From Tonkin to India by the sources of the Irrawadi," by Prince Henri d'Orleans. Translated by Hamley Bent, M.A., London, 1898. Also JOURNAL OF TROPICAL MEDICINE, February, 1900, p. 192.

² Quoted by Michoud. Yunnan-fu is a town of considerable size some 180 miles north of Mengtsz.

and Baber describe the strange behaviour of rats. "When these animals feel themselves ill," writes the former, "they come out in troops, rush into houses, run about madly, and, after twisting about on themselves, fall down dead; most often they die under the floor, where they give rise to an infectious smell, the cause of which is discovered too late." In regard to the larger animals, the most interesting view is that expressed by Mgr. Fénouil, who has stated that: "In the plains visited by *Yang-tzu-ping*, the first victim is invariably the rat whose snout is always close to the earth; in successive and regular order the pig, cat, dog and ox, and finally, man, whose mouth is most distant from the soil, are afterwards attacked."¹

This theory appears to have originated with the Chinese, who have long held that plague arises from the soil, and that it especially prevails after drought, because the cracks then found in the earth allow of the escape of a deadly miasm which causes the disease.² It is a natural corollary to this view to suppose that the nearer the earth the more concentrated is the poison, and that as the miasm may be supposed to slowly rise it attacks the small animals first, and successively those of larger and larger size. The view can, however, scarcely be accepted without caution, for not only has no similar occurrence been observed elsewhere, but some of the animals named, notably oxen, would appear to be immune to plague.

There can be little doubt that the disease in Yunnan is largely spread by the barbarous customs in vogue there of treating the sick and the dead. Michoud has given a harrowing description of their mode of disposal of the hopelessly sick. "On either side," he writes, "of that gate [the west gate of the city], we remember to have seen, in 1893, rows of human bodies leaning against the city wall, some still moving, others already stiffened by death—people dead or dying from plague. It is a custom of the Yunnanese parents or relations to remove patients in a hopeless state from their homes and even from the town and to let them die in the open air, as the superstitious Chinese fear that in case of death occurring in a house, the soul of the deceased may refuse to accompany the body, and remain to haunt the premises." Rocher, too, has described the Chinese mode of burial, which consists in placing the corpse on a bier and exposing it to the sun, with the result that, "anyone coming into the neighbourhood of these plague-stricken villages is almost asphyxiated by the nauseating smell which comes from these decomposing bodies."

The behaviour of the disease in and near Mengtsz in 1893 and 1894, immediately preceding its epidemic extension to Canton, Hong-Kong, and so to the rest of the world, is a matter of great interest. So far as can be gathered from the Chinese Customs Medical Reports plague was as active as usual in Mengtsz in 1893.³ It will be shown later that in that year it was more than usually active outside the Yunnan province,

and was already extending in the direction of Canton. In 1894 it was apparently particularly active in Mengtsz, where whole families were carried off by the disease, and as many as 25 deaths occurred in a single *yámen*.¹ In that year the epidemic lasted from May to August, and its course was the same as in preceding years; but in 1895 the disease was unusually late in appearing, and the outbreak lasted from July to September, instead of from May to July or August, as in previous years.² In that year the pest was more virulent than on any previous occasion. Finally, in 1897 and 1898, there was the usual annual epidemic, but in 1899 this was apparently absent.³

It will be convenient now to consider the spread of plague outside Mengtsz and the Yunnan province. Upon this matter Michoud writes as follows:—

"In northern Yunnan, at Yunnan-fu, the disease prevails chiefly in winter, but has lost nowadays its previous gravity. We are unable to explain this difference in the dates of appearance of *Yang-tzu-ping* in the north and south. The climate differs essentially in these two parts of the province. At Mengtsz the disease arises on the spot every year, whence, after penetrating every corner of the city it overspreads the whole plain, not sparing any village or hamlet, and frequently extending to Kwang-Si [the adjoining province on the east]. It has never reached Tonkin, although Mengtsz is but five days' journey from Laokai, the first French town on the Chinese frontier. Manhao, the first port on the Red River, half-way between Laokai and Mengtsz, and separated by only 40 miles from each, with which, moreover, it is in daily communication, is only exceptionally visited by *Yang-tzu-ping*, yet the cases recorded are said to have been imported from Mengtsz. Manhao lies some 3,600 feet lower than Mengtsz; although free from *Yang-tzu-ping*, it has a very bad name, owing to the prevalence there of the so-called mountain-fever. The muleteers from Mengtsz, as a rule, refuse to spend the night in that low-lying place, camp out on the mountains, and come down to the little town only in the daytime to load their boats. Nothing could prove better the influence of altitude on the development of *Yang-tzu-ping* than the immunity of that small place."⁴

At times the disease has spread outside the borders of the Yunnan province, and invaded not only that of Kwang-Si, as stated in the above passage, but also the province of Kwei-chow to the north-east and Kwang-tung (Canton) still further east than Kwang-Si.

In the Kwang-Si province is the important port of Pakhoi, on the shores of the gulf of Tong-King (or Tonkin), and it will be convenient here to trace briefly the course of the disease in that town. Plague, as far as is known, first appeared in Pakhoi about the year 1867.⁵ Its subsequent history is uncertain, but it appears to have been epidemic there each year from

¹ Quoted by Michoud.

² *Medical Reports*, No. 42. "The Chinese are of opinion that bubonic plague emanates from the ground, and is favoured by a long continuance of dry weather, when the earth becomes porous and numerous fissures appear on the surface, facilitating the escape of whatever causes the disease." This theory is almost as old as the history of the plague itself.

³ Michoud, *loc. cit.*

⁴ *Medical Reports*, No. 49, p. 8.

⁵ No. 50.

⁶ *JOURNAL OF TROPICAL MEDICINE*, February, 1900, p. 192.

⁷ This assumption as to the influence of altitude is difficult to maintain in view of the frequent spread of the disease to Pakhoi on the coast, and other low lying places mentioned later in the text.

⁸ Lowry, *Medical Reports of the Imperial Maritime Customs of China*, No. 24. Writing in 1882, he states in reference to the disease that "it occurred for the first time about 15 years ago."

1871 to 1877 and again in 1882. In the last-named year it began at the end of March and raged till June, killing, it was thought, some 400 or 500 persons in a population of about 25,000. In 1883 there was no outbreak;¹ but in 1884 it was again severe.² This epidemic also began in March, and appears to have lasted until June. From 1884 to 1894 Pakhoi was free from plague.³ In 1894 the disease broke out again in the spring, and was "more violent and fatal than in any previous year, at any rate since 1882." The British Consul there, in a letter to the Colonial Secretary, dated May 16, 1894, stated that already up to that date 300 or 400 persons were said to have died of plague.⁴ "Before 1875," he adds, "I learn from Chinese sources, only about 100 people had died in any year of plague; in 1884, 50 or 60 died;⁵ in 1891 about 40 or 50;⁶ but from February 14 to now, 300 or 400."

This renewed prevalence of plague in Pakhoi in 1894, after an absence of ten years is a fact of great interest. That year, as already pointed out, marked the commencement of the wide diffusion of plague which has characterised the last six years, and in that year the disease is found showing unusual and excessive activity both in and near its endemic home in China—the country from which the subsequent extension of the disease to the rest of the world is almost certainly to be traced.

In 1895 and 1896 no mention is made of Pakhoi in the Chinese Medical Reports; but in April and May, 1897, 20 deaths from plague occurred there, and there was a further slight recurrence in June, with a total of some 70 deaths.⁷ In the last issue of the Reports Pakhoi is again not mentioned.

Another important town, some 12 miles inland from Pakhoi, is that of Lung-Chou,⁸ which is of sufficient size to form a Chinese prefecture. Plague has frequently appeared here, sometimes in years when it has not spread to Pakhoi. In 1882 it began in March (as at Pakhoi) and raged until August.⁹ In 1883 it was severe here in May and June, and disappeared in July;¹⁰ in that year it was absent from Pakhoi, and there was no trace of it elsewhere in the province. In March, 1884, it again appeared in Lung-Chou, and possibly elsewhere in the province.¹¹ In the winter of 1885-6, a few cases of the disease occurred,¹² and again in 1890,¹³ in neither year spreading to Pakhoi. In 1893, 1894, 1895¹⁴ and 1896,¹⁵ the plague was present

in the town; in the last-named year it caused only a few isolated deaths. This appears to be the last mention of Lung-Chou in the Medical Reports.

That the infection of plague, when the disease breaks out in Lung-Chou or Pakhoi, is on some occasions imported there from Yunnan appears probable. In some years its importation has apparently been traced. Thus, in 1890 it is definitely stated that "having originated in Yunnan, it passed through the town of Po-sè, and the prefectural cities of Nan-Ning and Tai-ping in Kwangsi, and thence to Lung-Chou, also in Kwangsi."¹ Again, in 1893 and 1894, Simond² expressed the belief that plague was imported to a camp at Lien-Ch'eng, where an important collection of troops was stationed between Lung-Chou and the frontier, and that it was carried thence to the town, where the first cases appeared among the men of the garrison. In 1895, on the other hand, not a single soldier, either at Lien-Ch'eng or Lung-Chou was attacked. Elsewhere³ the same observer has stated that the infection was probably carried to Lung-Chou by mule caravans which are constantly passing between Upper Yunnan and the town. That it was so imported in 1893 is rendered probable by the fact that muleteers were among the first victims of the disease in Lung-Chou in that year. The following passage relating to this point is from the article by Michoud, already so freely quoted from:—

"In 1893, after having committed its usual ravages in Yunnan, the disease extended towards the south-east, and two months after its visit to Mengtsz we heard of its raging at Lung-Chou and in many parts of the Kwang-Si province. The commercial intercourse of Yunnan with Kwang-Si is incessant. Every day thousands of pedlars and numerous caravans of horses and mules are plying on the roads of the two provinces, evidencing the importance of the traffic existing between them—probable agents, too, of the propagation of such a contagious disease as the plague. It is worth noting that the disease does not remain permanently in the low regions of Kwang-Si and Kwang-Tung. The variability of its appearance in these places seems to point to a newly-imported contagion whenever the plague breaks out. One would think, in short, that in the low-lying regions the micro-organism of the plague does not find the conditions necessary to its vigour. On the Yunnanese highlands, on the contrary, if the disease is not perennial, it shows at least great regularity in its annual revival."

While this view of the annual or frequent importation of the infection to Lung-Chou and other places outside Yunnan has much in its favour, the possibility, on the other hand, cannot be denied that the recurrences in these places may be due to revival of infectious material remaining from preceding outbreaks. In other words, it cannot be positively asserted that plague is not endemic in Lung-Chou. In support of this view is the definite statement that the disease is endemic in other places outside Yunnan. Thus in a district near

¹ *Medical Reports*, No. 25 and No. 26.

² *Ibid.* No. 28. ³ Sharp Deane, *Ibid.* No. 47.

⁴ Parliamentary Paper, C. 7,461, correspondence relative to the outbreak of Bubonic Plague at Hong-Kong, 1894.

⁵ This is far below the estimate quoted above for the year 1884 (Lowry).

⁶ This is presumably an error, as Sharp Deane (*loc. cit.*) states emphatically that no plague occurred here between 1884 and 1894.

⁷ *Medical Reports*, No. 55.

⁸ This is frequently spelt Lien-Chou, or by French authors Long-Tchéou. So far as I can gather it is the same place which is referred to under these different guises. It is situated almost due north of Pakhoi, and in latitude 22° 25' N., and longitude 109° E. (approximately).

⁹ *Medical Reports*, No. 24.

¹⁰ No. 26. ¹¹ No. 28. ¹² No. 30. ¹³ No. 39.

¹⁴ No. 50. Delay, "*Rapport Médical pour l'année finissant le 30 Juin, 1895, sur la situation sanitaire de Lung-Chou.*"

¹⁵ No. 52.

¹ No. 39.

² Quoted by Delay, *loc. cit.*

³ *Archives de Médecine Navale*, 1895. "Notes d'Histoire Naturelle et Médicale Réceueillies à Long-tchéou," par L. P. Simond. Also, *Annales de l'Institut Pasteur*, 1898, p. 625.



AINHUM.

The 4th and 5th toes show the constrictions at their roots peculiar to the disease. The nails of the hands are affected by ringworm.



Keratosis of the feet in a case of Ainhum.



Keratosis of the hands in a case of Ainhum.

Photographs illustrating a case of Ainhum, described by Dr. A. B. DALGETTY, South Sylhet, India.

See page 193 of present issue.

An-p'u, about 100 miles to the east of Pakhoi (a town or village not marked on most of the ordinary maps of China) plague appears to be as truly endemic as it is in Yunnan. On this point Sharp Deane writes:—¹

"I am informed by one of the French missionaries who has resided for many years in the neighbourhood of An-p'u that bubonic plague is endemic in a small district near that place, and that isolated cases will be found there at any time of the year, but that during the early spring of some years the disease occurs as an epidemic, and then the only chance of escape is to leave the district until after heavy rain has fallen."

In March and April, 1893, plague carried off a large number of people in this district near An-p'u. The fact is of importance, as it shows that as early as 1893 a marked extension of plague activity was observed in the direction of Canton, where the disease appeared for the first time in history in the following year.

In the spring of 1894 plague broke out in Kao-Chao,² to the east both of Lung-Chou and Pakhoi. At the same time it caused a severe epidemic in Yang-Chiang,³ still further east, and still nearer to Canton. Both these places are in the Kwang-Tung province. In the summer of 1895 plague was severe at Kotak, a fishing port adjoining Pakhoi.⁴ It also attacked a few people at Hadung and at Thuy-Cao on the river Caobang.⁵

Before proceeding to discuss briefly the significance of these facts, it has to be pointed out that the Yunnan plague had apparently never spread to Tong-Kin or Annam before 1894, and the sole exception since that year has been the small outbreak, described by Yersin, of plague at Nha-Trang in 1898. That outbreak was of doubtful origin, but may have been due to an importation from Pakhoi or Hong-Kong.⁶ The provinces to which it has spread have, apparently, invariably been those only of Kwang-Si, Kwang-Tung (both on the southern coast of China), and perhaps occasionally Kwei-Chou, to the north-east of Yunnan. In former years it apparently sometimes spread to Laos,⁷ the region between Yunnan and the north of Siam.

To sum up briefly what is to be learnt from the evidence here brought together it may be stated that:—

1. In a region lying between 23° and 28° north latitude (bordering on and partly within the tropics) and between 100° and 104° east longitude plague has been endemic at least since 1860.

2. It may have prevailed here much longer, possibly for one or more centuries.

3. Its renewed prevalence after 1860 was associated with a rebellion and civil war.

4. It may have come from India through Tibet, but there is no positive proof that it did do so.

5. Within this area the course of the disease has been somewhat irregular; for, though in regard to

season it has shown marked regularity, it has not always attacked the same villages or town in each year. It has frequently passed over one or more to break out in a distant town or village.¹ Mengtsz and its neighbourhood have of recent years been the principal seat of the disease. It is much less severe now than it was in former years.

6. Plague has from time to time spread beyond the province of Yunnan. It is indeed, said to be endemic near An-p'u, one hundred miles east of Pakhoi; it may also be endemic in (or, if not, is frequently imported to) Lung-Chou.

7. It was epidemic in Pakhoi before and in the year 1877, in 1882 and 1884. In 1894, after ten years' absence, it reappeared here and caused a very violent epidemic.

8. In 1893 plague was as active as ever in the endemic area in and around Mengtsz. It raged in Lung-Chou and in many parts of the Kwang-Si province. It was unusually active in the endemic centre near An-p'u.

9. In 1894 plague appeared for the first time in history in Canton and Hong-Kong. Canton was first affected. The exact date of the appearance of plague there is uncertain—it was probably very early in the year, perhaps in January. In that year it was active, and apparently rather unusually active, in Mengtsz and neighbourhood; it raged at Lung-Chou. At Pakhoi (after a complete absence of ten years) it was more severe than in any previous year—at least since 1882. It broke out also at Kao-Chao and Yang-Chiang—places in the same province as Canton and between Lung-Chou and that city. A few cases also occurred at Hadung and Thuy-Cao. None of these places had been previously mentioned as affected by plague, with the exception of Kao-Chao in 1891.

10. In 1895 it raged at a later period than usual, but was more virulent than it had ever been known to be in Mengtsz.

Finally, from these collected observations, it would appear that in the year preceding the epidemic extension of plague to Canton and Hong-Kong, which proved to be the commencement of an almost world-wide extension of the disease, plague was unusually active in its endemo-epidemic home in China. In the year of its epidemic extension to the places named, it was even more unusually active, spreading to several places where it had either never been before or had not been for the preceding ten years. In the following year its virulence in its great endemic home in Mengtsz was greater than it had ever been known to be in previous years.

In subsequent papers it will be shown that the other endemic centres of plague on the earth's surface were not showing any unusual activity at this time. It may, therefore, be asserted, with some confidence, that the epidemic extension of plague which marked the year 1894 and subsequent years, was directly due to a spread of infection from the endemo-epidemic centres of the disease in Southern China, and not to any universal or earth change simultaneously affecting the centres of the disease at distant parts of the earth's surface.

¹ *Medical Reports*, No. 45.

² No. 48. It should be noted that plague had also appeared in Kao-Chao in 1891. *Medical Reports*, No. 42.

³ *Ibid.*

⁴ No. 50, p. 32.

⁵ *Ibid.*

⁶ *Annales de l'Institut Pasteur*, March, 1899.

⁷ Rocher (*op. cit.*) writes: "At times it rages in Laos and on the frontier of Kwei-Chou."

¹ Roche, *loc. cit.*

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THE

Journal of Tropical Medicine

MARCH, 1900.

THE REPORT OF THE PLAGUE COMMISSION ON HAFKINE'S PROPHYLACTIC.

THE Commission which were appointed to inquire into the plague in India have issued their report on their investigation into Hafkine's inoculations. The system is subjected to a severe criticism, an air of scepticism prevailing throughout the report, which is far from indicating any bias in favour of either the methods or the results. Objections are made to the methods of standardising and storing the prophylactic, to the variation in doses, to the employment of the supernatant fluid, and to some of the statistical methods of stating the results. An analysis of the objections shows them, however, to be of less importance than

the Commission would lead one to believe by the emphatic manner in which they are put forward. The Commission themselves, after stating all that can possibly be brought against the inoculation, declare just as emphatically that the inoculations are harmless, that they greatly diminish the plague death rate among the inoculated, and that they sensibly diminish the incidence of plague attacks on the inoculated population. These are very important conclusions, far outweighing any of the opposing but unsupported theories which are to be found in various parts of the report. It is clear that the inoculations under present arrangements are a success, and whatever improvements are to be made on them can only be with the object of making them a greater success. Everyone will admit that the methods of standardising and of storing are capable of improvement. Such a criticism applies to most methods. It is only recently that ordinary vaccine has been protected from adventitious germs by the process of admixture with glycerine, but though it is an improved method and will add to the success of vaccination, yet it does not affect the proved efficacy of the vaccine used prior to this innovation. The defects in the preparation and storage of the plague prophylactic lie in a badly equipped laboratory, want of scientific assistants, and the extraordinary demand for an immense quantity of the prophylactic in consequence of exclusive plague epidemics. We doubt whether the true cause of the defects has been sufficiently appreciated by the Commission, for we do not see any mention in any part of the report of the importance of having for such work an adequate and scientific staff. It seems to be forgotten that there is nothing in the laboratories in England, or, indeed, in Europe, to be compared with the out-turn of the plague prophylactic in Bombay, and that there is no laboratory of any importance in Europe so badly equipped and understaffed with scientifically-trained men. Under these circumstances, to expect ideal standards which are not even attained in England in regard to diphtheria serum, nor even in the most recent attempts at standard-

ising the typhoid prophylactic is, to say the least, not a particularly sympathetic attitude, but one which is unfair to Mr. Haffkine and his system. It is perfectly marvellous that Mr. Haffkine should have been able to have accomplished so much with the passive opposition which he has encountered, and with the appliances and assistance which have been so grudgingly given him. A trace of that desire to hamper rather than to encourage is unfortunately not altogether absent from the Commissioners' report. We can view the theoretical criticism of the composition of the prophylactic in no other light, for there is not the slightest evidence put forward by the Commission to justify them arriving at the opinion that there is no efficacy in the supernatant fluid. They seem here to be rather the advocates of another system than inquirers into the present system, and have unwittingly been led astray. As a matter of fact, against the experiments they mention there are later ones which show that the supernatant fluid possesses protective properties. We have only to mention those of Dr. Balfour Stewart, which we understand have been fully confirmed by Surgeon-Captain Milne. Similarly, the statistical method of statement adopted by the Commission minimises the effect of the inoculation to an extent that is misleading. They adopt a method of statistical expression which is not admissible, subtracting percentages and treating the results as the difference between the mortality of the inoculated and uninoculated. Thus, for Hubli they give the following figures:—

		Average Strength.		Number of Plague Deaths.		Percentage of Deaths on Average Strength.
Uninoculated..	..	20,225	..	980	..	4·8
Inoculated	22,967	..	118	..	·5
Diminution of mortality in favour of inoculated, 4·3.						

The figure 4·3 is altogether misleading; the ratio is actually 9 to 1, or, in other words, the uninoculated died at nine times the rate of the inoculated. By subtracting percentages a figure is obtained which will not bear comparison with figures obtained in a similar fashion. Thus, if 99 per cent. is subtracted from 100 per

cent., 1 per cent. is the result. Similarly, if 1 per cent. is subtracted from 2 per cent., the result also is 1 per cent. Yet neither of these results, though apparently the same, are any where near the same value—in one case representing a hundredth part, and in the other the half of a whole. The Commission are entirely at fault with their statistics, and, no doubt, on this account are unable to give an arithmetical estimate of the value of the inoculation. It would have been better for them to have adhered to the form adopted by Mr. Haffkine, which is obtained, unlike that of the Commission, by the correct and ordinary rules of statistics. This error is so manifest that it causes some doubt as to whether the criticism on the statistics is entitled to the full amount of confidence that it would otherwise receive. But even when accepted to its fullest extent, the inoculations remain unshaken, and the Commissioners recommend that, under the safeguard of accurate standardisation and precautions for proper sterilisation, the inoculations should be encouraged wherever possible, and in particular among disinfecting staffs and the attendants of plague hospitals.

MALARIOLOGY.

DR. MANSON, C.M.G., addressed the members of the Royal Colonial Institute on March 13, 1900, on "A School of Tropical Medicine." Sir Henry T. Jourdain occupied the chair. Dr. Manson dealt with the subject of the teaching of tropical diseases as a branch of public health, and showed most lucidly and effectively the benefit likely to accrue from a more intimate knowledge of the diseases of the tropics. He desired not only to instruct members of the medical profession, but he believed that it is of immense importance to inform the public of the problems in hand, so that they may be willing to accept them and to co-operate in their practical application. Dr. Manson referred to malaria as the principal cause of sickness and death in the tropics, and of social stagnation: "It is the king there, and like some brutal tyrant it blasts its

subjects and its kingdom." After referring in general terms to the enormous mortality in India, where some 5,000,000 perish annually from fevers, mostly of a malarial type, Dr. Manson gave a concrete example of the mortality, the expense and the dislocation of trade and government by malaria in the Gold Coast Colony. It is satisfactory to learn that the Colonial Office has authorised Dr. Manson, in conjunction with the School of Tropical Medicine, to make two experiments as to the practicability of preventing malaria in malarial localities by practicable and easily applied means.

Experiment 1:—"Such a hut as would be suitable for the European to live in in tropical Africa, is to be erected in the most malarial part of the Roman Campagna available. The hut is to be furnished with wire gauze door and window screens and other devices to render it mosquito proof. Two skilled observers and their two servants are to live in this hut from May till October of this year—that is, during the entire malarial season. Of course they will be at liberty to go where they like during the day, but from an hour before sunset to an hour after sunrise they are to be in the hut. Now, if these men escape from fever it will be absolute proof that by very simple and inexpensive means the human body can be protected from the malaria germ, for, as regards the spot in the Roman Campagna selected for the experiment, I may state that to sleep there unprotected but for one night is regarded by the Romans as tantamount to contracting a malarial fever, and that, too, of a virulent type."

Experiment 2 is to be as follows:—"Laboratory bred mosquitoes, that is, mosquitoes raised from the egg in the laboratory, and which have had no opportunity of picking up malarial germs in the haunts of malaria, are to be fed in Rome on patients in whose blood the benign tertian malaria parasite has been ascertained by the microscope to be present. These mosquitoes are to be transported to London, fed on vegetable juices till such time as we know that the malaria germs shall have arrived at the venom gland. The insects are then to be liberated in a small mosquito house in which one or more English-

men who have never left this country are to sleep. We expect, that in about ten days after this, these Englishmen will develop malarial fever, and that we shall find the malaria parasite in their blood. There is no danger from this experiment, as the type of malaria parasite we propose experimenting with is not virulent and is easily killed by quinine."

Although, as Dr. Manson says, neither of these experiments are new, so that for the malariologist positive results are a foregone conclusion, they have never been combined in the crucial way proposed.

THE PRACTICAL DETAILS CONNECTED WITH VACCINATION AGAINST TYPHOID (ENTERIC) FEVER.

Directions issued with PROFESSOR WRIGHT'S Anti-typhoid Vaccine Tubes.

I.—PRINCIPLES UPON WHICH THE ANTI-TYPHOID INOCULATIONS ARE BASED.

It has been firmly established by laboratory experiments that animals which have been inoculated with dead cultures of typhoid bacilli possess, as compared with normal animals, an increased power of resisting infection by living typhoid bacilli.

It may be inferred with probability from these experiments that inoculations with dead typhoid bacilli will, in like manner, confer upon man an increased power of resisting typhoid infection.

This expectation is further justified by two facts. First, it is justified by the fact that the anti-typhoid inoculations induce in man precisely the same blood changes which they induce in animals. Further, it is justified by the fact that the blood changes, which are induced in man by the anti-typhoid inoculations, are precisely the same as those which are induced in him by an actual attack of typhoid fever. The importance of this last fact becomes clear when it is considered that the insusceptibility against further attack which supervenes upon an attack of typhoid fever is almost certainly dependent upon the recurrence of the particular blood changes which are here in question.

II.—COMPOSITION AND METHOD OF PUTTING UP THE ANTI-TYPHOID VACCINE.

The vaccines consist of sterilised typhoid cultures. These cultures have in some cases been grown on agar-agar, but, as a rule, they have been grown in nutrient broth for from three to four weeks.

The vaccines are put up—(a) in sealed glass capsules, (b) in small glass bottles which are covered by paraffined india-rubber caps.

The vaccine which is put up in glass capsules is to be employed when only one or two vaccinations are undertaken at the same time. The vaccine which is contained in the bottles is to be employed

when a larger series of vaccinations are to be undertaken.

After the sterility of each sample of vaccine has been determined, a small quantity of antiseptic is added with the view to securing it against the possibility of subsequent contamination.

III.—METHOD OF DRAWING OFF AND OF INJECTING THE ANTI-TYPHOID VACCINE.

(1) *Method of Sterilising the Hypodermic Syringe.*—The hypodermic syringe may be most conveniently sterilised by filling it in with sweet oil,¹ which has been brought to the temperature of 140° to 160° C. The oil may be heated over a spirit lamp in any shallow vessel (in default of a better vessel it may be heated in a table-spoon). A piece of ordinary bread crumb serves as a useful thermometer. The point at which a sufficiently high temperature is attained is indicated by bread *beginning* to turn brown. When this temperature has been attained² the hot oil is drawn up into the syringe. Sterilisation will be complete as soon as the hot fluid has come into contact with the whole of the interior of the barrel.

Lastly, the needle is to be sterilised by dipping it into the hot oil. This done, it is to be firmly fixed down upon the nozzle of the sterilised syringe.

(2) *Method of drawing off the contents of the Vaccine Capsules.*—First thoroughly shake up the contents of the capsule, so as to bring into suspension the bacterial deposit which may be adhering to the walls of the capsule. Then sterilise the tip of the capsule in the flame. This done, carefully break off this tip with a pair of sterilised forceps, or better, cut it off with a pair of sterilised scissors. Now hold the sterilised syringe needle upwards and invert the capsule over the point of the needle. The contents of the capsule may now be drawn off aseptically into the syringe. While drawing out the piston of the syringe, care must be taken to keep the point of the hypodermic needle continuously below the surface of the fluid in the capsule.

(3) *Method of drawing off the Vaccine from the Rubber-capped Bottles.*—First thoroughly shake up the contents of the bottle, so as to bring into suspension the bacterial deposit which will have accumulated on the floor or sides of the bottle. Then melt off the paraffin and sterilise the surface of the cap by dipping it into a boiling 5 per cent. solution of carbolic acid. This done, fix the bottle mouth downwards in a clamp³ at a convenient height above the table. Now pierce the rubber cap with the sterilised needle of the hypodermic syringe and withdraw the needle.

¹ It will be obvious that the advantage which is obtained by the substitution of oil for water as a sterilising agent, consists in the fact that, owing to the higher boiling point of oil, a temperature higher than that of 100° C. can be brought to bear upon the articles which are to be sterilised. Another advantage which is incidental to the use of the oil is the preservation of the hypodermic needles from rust.

² If the plunger, or any other of the fittings of the syringe, is made of india-rubber, care must be taken to see that the temperature of the oil or glycerine does not rise beyond the point indicated above.

³ Where a retort stand and clamp are not available, the bottle of vaccine may be simply laid on its side on a table or held by an assistant.

This first puncture, be it noted, is to serve only as an air-hole. Having now made provision for the entrance of air, pass in the needle again into the bottle through another portion of the rubber cap. The vaccine can now⁴ be drawn off into the syringe.

When the contents of the syringe have been used up, and the syringe is to be filled in again, the needle must be resterilised before it is reintroduced into the bottle of vaccine. The necessary sterilisation may be effected by merely dipping the needle as before into the superheated oil.

When the series of inoculations has been completed, the surface of the rubber cap is to be flushed with spirit. On the evaporation of this spirit, the punctures in the rubber cap may be closed either by immersing the whole rubber cap into a vessel containing very hot melted⁵ paraffin, or by coating over the surface of the cap with some dissolved rubber solution. In any case, where the vaccine is to be employed again, the greatest care must be exercised to see that it is sealed up air-tight.

(4) *Instructions as to the method of Injecting the Vaccine.*

(a) *Choice of a site for the Injection.*—With a view to diminishing the pain which is consequent upon serous effusion, and with a view to facilitating the absorption of such serous effusion, it is advisable to inoculate into the flank, where the subcutaneous tissue is loose, rather than into the arm where the skin is more tightly tied down to the underlying tissues.

These considerations are of especial importance in dealing with lymphatic persons, in whom a not inconsiderable amount of effusion is prone to occur.

(b) *Method of making the Injection.*—The most convenient method of making the injection is to pick up a thick fold of skin between the finger and thumb, and then to pass the needle well down into the subcutaneous tissue in the centre of this fold.

In every case it is advisable to wash the skin with an antiseptic solution before inserting the needle.

(c) *Additional Instructions as to the procedure which is to be adopted when undertaking a series of Inoculations.*—When inoculating a number of persons in series it will be a matter of convenience to employ a 5 cc. syringe, which will contain sufficient vaccine for some five to ten inoculations. Further consideration will show that, inasmuch as nothing can pass into the interior of the syringe while the inoculations are proceeding, the interior of the syringe will not require to be resterilised after each separate inoculation. It will therefore suffice to ensure the antisepticity of the whole proceedings if the hypodermic needle be resterilised after the completion of each separate

⁴ Difficulties in drawing off the vaccine can only occur in cases where the tip of needle becomes blocked with paraffin. This difficulty, which will not occur if the instructions as to removing the paraffin are carefully carried out, can easily be remedied by passing the tip of the needle through the flame.

⁵ The hard paraffin which is employed in laboratories is the most suitable for this purpose. Where this is not available, a piece of ordinary paraffin candle may be melted down with a drop or two of sweet oil. The india-rubber solution which is used for mending bicycle tyres may be employed for this purpose after the orifice of the collapsible tube has been flamed. Frequent experiment has shown that the solution itself may be taken as sterile.

inoculation. This, like the re sterilisation of the needle, which must always be undertaken before the syringe is reintroduced into the bottle of vaccine, may most conveniently be effected by merely immersing the needle after each inoculation in a vessel of oil which is kept at a temperature of over 100° C.

IV.—DOSAGE.¹

The dose of vaccine which is to be administered is in every case specified upon the bottles and capsules. It is to be understood that the dose which is specified is the dose which is applicable to an ordinary male adult. In dealing with women, children, or with persons of feeble physique, the dose is to be proportionately reduced. Further, it is to be understood that the dose, as specified on the capsules and bottles, applies only to the first vaccination. Where a second vaccination is undertaken, within ten days or a fortnight after the first vaccination, one and a half to twice the original dose may be administered. In this case the operator will do well to guide himself in the selection of his dose by the severity or otherwise of the symptoms which have been produced by the first vaccination.

V.—CLINICAL SYMPTOMS WHICH MAY BE EXPECTED TO RESULT FROM THE ANTI-TYPHOID INOCULATION.

The clinical symptoms which result from anti-typoid inoculations are subject to considerable individual variations.

(1) *Constitutional Symptoms.*—Some degree of malaise and some slight tendency to faintness may be expected in every case. In a very small percentage of cases there may be a definite rigor or even a certain amount of collapse. These symptoms may be expected between the first and the sixth hour. Where they are at all severe it is the rule for them to come on before the expiration of the third hour.

These preliminary symptoms are followed by a certain amount of fever. The average temperature which is attained is about 101° F. In exceptional cases the fever may go as high as 103° F. The fever generally passes off completely at the end of eighteen to twenty-four hours, but it may, in exceptional cases, persist for another twenty-four hours.

(2) *Local Symptoms.*—In every case a certain amount of local tenderness will develop. This will generally begin to make itself felt about five to six hours after the inoculation. Somewhat later a red blush will appear around the site of inoculation. The local tenderness will be at its worst in about eighteen hours. In many cases the skin will then be red over an area of four to five inches square and lines of injected lymphatics will often be traceable upwards into the armpit (along the border of the pectoralis

major), and downwards into the groin. There may in addition be slight tenderness in the groin or armpit. These local symptoms will have practically passed away after the expiration of forty-eight hours.

In some cases where the constitutional symptoms are severe, there is an almost complete absence of local reaction.

VI.—SUGGESTIONS AS TO THE TREATMENT WHICH MAY BE ADOPTED FOR THE ALLEVIATION OF THE CLINICAL SYMPTOMS WHICH SUPERVENE UPON THE INOCULATION.

The most important point which has to be attended to after the inoculation is the possibility of the super-vention of faintness. This may be best guarded against by enforcing upon the patient the necessity of remaining as quiet as possible after the inoculation. Exposure to the sun in tropical countries and violent exercise are at any rate to be carefully avoided. These ends are, in the case of soldiers and others, most easily achieved by choosing the late afternoon as the time for the performance of the inoculations. The patients are to be enjoined to go and lie down as soon as they feel at all ill.

It is best not to interfere in any way with the fever which supervenes.

In connection with the alleviation of the local swelling and tenderness, it will be found that the tendency to serous effusion will be greatest in persons whose blood coagulability is naturally abnormally low. The serous effusion can in such persons be held in check by the administration of 30 grains of calcium chloride, *cryst.* This dose may be administered at the time of inoculation, and may be repeated six to twelve hours afterwards. The local tenderness and stiffness which supervene from the inoculation may always be sensibly relieved by the application of hot water stupes.

VII.—REMARKS ON THE QUESTION OF THE ADVISABILITY OF SECOND INOCULATIONS.

Comparative observations on the condition of the blood in once and twice inoculated patients have shown in the most distinct manner that the antidotal substances which are produced in the blood by the first inoculation are considerably increased under the influence of a second inoculation.

There can therefore be no doubt that a patient will always reap considerable additional protection from a repetition of the inoculation.

VIII.—DATE AT WHICH THE SECOND INOCULATION OUGHT TO BE UNDERTAKEN.

It is well in all cases to postpone the second inoculation until eight or ten days have elapsed from the date of the first inoculation. In other words, it is well to abstain from introducing further typhoid toxin into a patient until his system has completely neutralised the toxin which was introduced at the first inoculation.

IX.—METHOD OF TESTING THE EFFECT OF AN ANTI-TYPHOID VACCINATION.

The method of testing the result of an anti-typoid vaccination has been fully described in a series of papers which have emanated from the Pathological

¹ The dose which is prescribed on the bottles is arrived at by determining the quantum of vaccine which is lethal for a guinea-pig of 250 grms. Two-fifths of this lethal dose constitutes, in the case of the vaccine which has been grown upon nutrient broth, an adequate dose for an adult man. In the case of the vaccine which has been grown upon nutrient agar the prescribed dose corresponds to a smaller fraction of the afore-mentioned lethal dose. In almost every case, but especially in the vaccine which has been grown upon agar, the data which are obtained upon guinea-pigs have been further checked by a direct observation of the effects of the vaccine upon man.

Laboratory of the Army Medical School. These papers were published in the *British Medical Journal* of January 30, 1897, May 15, 1897, and February 5, 1898; and in the *Lancet* of March 6, 1897. In its essentials the method of testing is as follows:—A small quantity of blood is withdrawn from a prick in the finger. The serum which is obtained from this blood is diluted 5, 25, 50, and 100 fold. Each of these dilutions is mixed with its own volume of a recently prepared dead typhoid emulsion. If the vaccine has produced its desired effect upon the patient's blood, the phenomena of agglutination and sedimentation will be observed to occur in each of these serum dilutions. In other words, the patient's serum will, in a 10, 50, 100, and 200 fold dilution, be found to exert a chemical effect upon the protoplasm of the typhoid bacteria.

X.—QUESTION AS TO THE DURATION OF THE IMMUNITY WHICH IS CONFERRED BY THE ANTI-TYPHOID INOCULATIONS.

Two entirely distinct series of data will ultimately be available for the solution of this question. On the one hand it will probably be possible to determine the duration of immunity by making observations on blood which has been drawn off from vaccinated persons at various intervals after their last inoculation. For it may be taken as probable that the protection persists as long as the antidotal substances, which are produced by the inoculation, are to be found in the blood. On the other hand, it will clearly be possible, in cases where large and compact bodies of inoculated men are continuously exposed to typhoid infection, to infer the persistence of the protection from their continued immunity from attack. In cases of this sort the proof of continued protection will of course be incomplete, unless typhoid is occurring in uninoculated persons who are living under the same conditions.

Owing to the fact that anti-typhoid inoculations have been only so recently introduced, and owing also to the difficulties which are associated with the collection of data from inoculated persons who are scattered all over the world, it has not yet been possible to collect sufficient statistics to give any definite answer to the question as to the duration of the immunity.

Such few data as have already been collected may be summarised as follows:—

Blood examinations which have been made, some at an interval of one year, and others at an interval of two years after inoculation, have shown that, in a large percentage of cases, the antidotal substances persist, in diminished but still very sensible quantities, for a minimum of one to two years after inoculation. Sufficient time has not yet elapsed to permit of an answer to the question as to whether the antidotal substances in the blood persist for a period exceeding two years.

Further, such reports as have been received regarding the continued immunity of the inoculated seem, so far as they go, to warrant the conclusion that a protective influence persists for a minimum of one to two years.

XI.—KEEPING OF RECORDS AND COLLECTION OF DATA WITH RESPECT TO THE EFFICACY OR OTHERWISE OF ANTI-TYPHOID VACCINATIONS.

It is earnestly requested that, in every case a record

of the inoculations which have been performed may be sent with the least possible delay to the undersigned. These records should specify the name, age, and occupation or rank of the inoculated persons.

Further, it is earnestly requested that any evil effects which are observed to supervene upon the injection of the vaccine, or any facts which throw any doubt on the efficacy of the vaccine, may be immediately reported.

Lastly, it is requested that any facts which go to show that protection is afforded by these inoculations may be similarly reported. In cases where the inoculated have escaped attack, while the uninoculated have been attacked, it is requested that the names of the inoculated and uninoculated shall in all cases be given, and that any differences which may have existed between the conditions of life of the inoculated and uninoculated may be fully pointed out.

In collecting data with regard to the effect of the anti-typhoid inoculations, attention ought to be further directed to the following points:—

(a) *The anti-typhoid inoculations will do nothing to check an incipient attack of typhoid fever.*

The fact that the anti-typhoid inoculations do nothing to check an incipient attack of typhoid fever must be borne in mind whenever these inoculations are undertaken in the actual presence of a typhoid epidemic. For, under such circumstances, it will every now and then happen that a man will present himself for inoculation when he is already in the incubation stage of the fever. If such a man is inoculated, the fever which he is incubating may be expected to run its ordinary course.

(b) *The protection which is conferred by the anti-typhoid inoculations is probably not established till after the expiration of 3—5 days.*

This consideration has an obvious importance in view of the fact that a man who is continuously exposed to the typhoid infection may possibly take in that infection during the first few days after inoculation. Cases of this kind, if they can be shown actually to occur, obviously ought not to be imputed to any defect in the vaccine.

(c) *The blood of patients who have been inoculated against typhoid gives Vidal's reaction, just in the same way as the blood of a patient who is suffering from an actual attack of typhoid fever.*

This fact is of importance in connection with the occurrence of cases of continued fever in persons who have been previously inoculated. For it is to be kept in view that Vidal's reaction, when it is obtained in a person who has been previously inoculated, does not in any way confirm the diagnosis of typhoid, which may have been arrived at by ordinary clinical methods. Much less does it conclusively prove the correctness of such diagnosis.

In view of the difficulty which will therefore often arise in connection with the recognition of typhoid in inoculated persons, it is manifest that *the effect of the anti-typhoid inoculations will be more accurately gauged by the reduction of the actual typhoid mortality, than by the reduction of the number of presumed cases of typhoid fever.*

XII.—METHOD OF OBTAINING THE ANTI-TYPHOID VACCINE.

Applications for anti-typhoid vaccine may be addressed to the undersigned,

A. E. WRIGHT, M.D.,
Professor of Pathology,
Army Medical School, Netley.
Pathological Laboratory, Netley.

British Medical Association.

SUPRAHEPATIC ABSCESS.

By JAMES CANTLIE, M.B., F.R.C.S.
Surgeon to the Seaman's Hospital Society.
(Continued from p. 191.)

SIGNS AND SYMPTOMS.

In addition to the ordinary symptoms attendant upon febrile disturbance we have the local sign of effusion in the right subdiaphragmatic, or, as I prefer to call it, the suprahepatic region. Pain in the early stages is caused by the mobility of the inflamed parts. The patient at first has not learned to accommodate the chest movements so as to ameliorate the pain; but soon the chest movements are adapted by the breathing becoming costal, so that the attachments of the diaphragm are left at rest as much as possible. The pain is of a dragging character, the dial of pain is to be found within the right acromio-clavicular angle. When the upper surface of the diaphragm becomes involved in the inflammation, the pleural surface opposite the base of the right lung and the visceral layer covering the right lung base itself are inflamed, giving rise to sharp twinges of pain. This lasts but a short time, however, and may be merely transient owing to the speedy adhesions that form between the pleural surfaces. When pus has actually formed, there is frequently no pain complained of, and the patient may divert attention from his liver by some pulmonary, gastric, intestinal, or feverish ailment, which, whilst dependent upon the formation of pus, serves to obscure its presence.

LOCAL SYMPTOMS.

Percussion and palpation of the liver, made in the usual way, may elicit neither tenderness, enlargement, nor abnormality of margin or surface. If, however, one hand is placed behind over the right lower ribs and the right loin, whilst the other hand is laid on the anterior wall of the abdomen just below the right costal arch, and the liver grasped firmly at the same time causing a to-and-fro motion of the liver, a sharp, shooting pain is immediately felt in the right shoulder at the acromio-clavicular angle. The pain thus elicited is of so intense a character that the patient dreads any attempt at a repetition of the examination, and prays to be spared the agony. Whilst percussion of the lower limit of the liver affords well nigh negative evidence, it is different on the upper aspect. Percussion above the right hepato-pulmonary line reveals an increase in dullness upwards. The dull area,

moreover, is characteristic in shape and fairly constant in position. In the neighbourhood of the right nipple, whilst percussing from the sternum outwards, the area of dullness is found to rise suddenly and drop behind as suddenly, mapping out a rather conical-shaped patch, with its base below at the liver and its apex above, usually a little within the nipple line. The cone may be fairly sharp, or it may be blunted, the dull patch may in fact present an outline like an inverted saucer, or even a bowl on the top of the ordinary line of hepatic dullness. The intercostal spaces over the dull area may bulge, and percussion of these spaces may or may not be attended by pain.

It frequently happens that the lower lobe of the right lung becomes congested. The congestion is what might be termed a fleeting congestion, for whilst one day the liver dullness (the dullness caused by the area of the pus), and the pulmonary dullness due to congestion, are indistinguishable, on the next day the lung may have cleared up, leaving the cone of dullness, due to pus, distinctly mapped out. These lung lesions vary from day to day, and I have been frequently deluded into the hope that I was wrong in my diagnosis, and that what was the matter was but a right pulmonary congestion after all, and not a serious hepatic trouble. These fleeting lung congestions and clearings are apt to deceive the beginner and to cause him, as it has caused me, to delay operation until it is too late.

It is unnecessary to go over the other well-known clinical signs and symptoms—the characteristic cough, the pain induced by lying on the left side, the alternating constipation and diarrhoea, the accompanying fever, the night sweats, and many other minor ailments which for the most part come and go.

I must remark, however, upon the absence of pain and rise of temperature. Neither pain nor increase in temperature are necessary adjuncts to the presence of pus in the region of the liver. When the initial attack of fever is over there may be no more evidence, except by clinical examination, that there is any collection of pus in or around the liver. I remember a patient of Inspector-General Turnbull's, in Hong Kong, who had had neither pain nor any increase of temperature for fourteen days before operation, yet a couple of pints of purulent fluid were withdrawn from a conical-shaped area of dullness on the top of the liver in the neighbourhood of the right nipple.

DIAGNOSIS.

When in warm climates a right lung basal congestion suddenly develops, trouble in the hepatic region ought to be suspected. When the congestion is frequent and recurrent, when the hepatic dullness along the upper border rises in a conical shape in the neighbourhood of the right nipple, when the liver itself is but slightly or not at all painful or enlarged, but when the liver is grasped and moved to and from a sharp lightning pain shoots up to the right shoulder, a suprahepatic inflammation may with fair certainty be assumed to be present. The needle of the aspirator, however, is the ultimate arbitrator in the question of diagnosis.

PROGNOSIS.

By early treatment—that is, aspiration and subsequent tapping and drainage—the mortality from liver

abscesses has been greatly reduced. Of 30 cases of liver abscess, on which I have either operated or been present at the operation, 4 died and 26 recovered. Of these, Inspector-General Turnbull, R.N., had 3 operations and 3 recoveries; Dr. Alex. Cowie had 4 operations and 4 recoveries; Dr. Stedman had 3 operations and 2 recoveries. Of 20 cases on which I operated 17 recovered and 3 died. Two of my 20 cases were the first two cases of liver abscess I ever saw or treated, and they were lost by delay. Both ultimately coughed up the hepatic pus, which is considered the right thing to hope for by many who pursue an expectant treatment. I know many cases recover after the pus has burst upwards through the liver, but it is a risky line of treatment to pursue, and I have come almost to this conclusion, that a practitioner who allows a hepatic abscess to burst upwards through the lung has not done his duty by his patient.

TREATMENT.

There is but one line of treatment to pursue in suprahepatic, or in intrahepatic, inflammatory conditions which threaten to develop pus. It is to aspirate early, to tap by trocar and cannula if pus is found, and to drain the abscess. Aspiration is almost without danger, but it is necessary to have a specially long needle made for the purpose. It should be about six inches in length, so that you can reach the most remote parts of the hepatic area, if so deep a puncture is necessary. Again and again it may be necessary to put the needle into the liver, either by changing direction or by separate punctures, and every probing does good. The abscess cavity may, however, be missed, and the chief cause of this is the assumption that the pus is in the liver. Had I acted on these premisses, eight men I know of would have in all probability died. The abscess has to be reached through the chest wall from the back, sides, or front. When far advanced the pus will be found bulging forwards in the area of the nipple line, and may there be found; but in earlier stages suprahepatic pus may be approached from the side, entering the aspirating needle in the mid-axillary or anterior axillary line between the seventh and eighth, or eight and ninth ribs.

I have stated that every additional puncture of the liver, made during examination, is in all probability attended by relief. What happens to the liver when it is punctured, and how can good arise? I am able to afford some explanation of this phenomenon. Whilst tapping a man for ascites dependent on liver enlargement, I pushed an aspirating needle into his liver in two or three places for the purpose of clearing up my diagnosis. On withdrawing the aspirating needle, the ascitic fluid which was previously perfectly clear, became tinged with blood. As the fluid flowed, it became more and more sanguinous, colouring the ascitic fluid deeply. The patient was greatly relieved by the combined operation, whilst I gained the knowledge that the puncture caused by the aspirating needle bled freely, thereby accounting for the immense relief always bestowed upon patients suffering from hepatitis by puncturing the liver. I am convinced that were the aspirator resorted to in the early stages of hepatitis, there would be fewer

liver abscesses. To do good the operation must be done early, and instead of losing time by deluging the liver with antiphlogistic drugs and applying counter-irritants, it is a safe, reliable and speedy method of relieving hepatic inflammation to draw off blood by an aspirating needle early in the disease.

TAPPING.

When pus is found the abscess must be immediately tapped by a trocar and cannula. There are many advocates of incision. The old "operations," as they were termed, for liver abscess consisted of waiting patiently until the abscess bulged at the abdominal wall or through the ribs, when a cutaneous incision was all that was necessary to reach the pus. Still these were dignified by the name of "operations for liver abscess." But another style of operator has arisen who removes pieces of ribs, sews up the pleura, makes an opening in the diaphragm, stitches the parts together so as to make a channel for the pus to travel. I was shown the use of the trocar and cannula by Dr. Manson, and I have used it on many patients and never had occasion to regret it. I followed the principles he taught, but modified in some degree the practice. As it is important that one should know how deep the needle penetrates, and how far the trocar and cannula ought to go, I had both needle and cannula marked in inches, so that it is possible to know exactly how deep one has punctured, and how far to push in the trocar and cannula after withdrawing the needle. After plunging a large trocar along the tract of the aspirating needle, and as far as the needle went, I withdraw the trocar, leaving the cannula. Through the cannula left in position an indiarubber tube of a size considerably larger than the cannula is introduced by being stretched on a metal rod. The metal rod carrying the stretched tube is introduced to the bottom of the wound, the cannula slipped over the tubing, and then, with the rod held against the bottom of the cavity, the tube is relaxed and the rod withdrawn. The piece of tubing may be cut short off by the skin wound, or into the projecting end a piece of glass tube three or four inches long may be inserted, and a drainage tube conducted therefrom over the edge of the bed to a basin containing antiseptic fluid. I dispense with Dr. Manson's rather complicated and elaborate dressing and apparatus calculated to prevent infection of the wound by entrance of air. The tissues from the skin to the pus which have been pushed inside by the bayonet-pointed trocar do not lose their elasticity, but embrace the indiarubber holding it firmly in position. My method of introducing the indiarubber tubing is simple in the extreme, merely catching the rubber in a projecting spike from the rod. By this means the end of the tube is left open, a most important point in my estimation, and one which leads me to dispense with Dr. Manson's method of drainage in which the end is closed, the pus having to find its way through the eyelets cut in the sides of the tube. No doubt Manson's drainage answered in large abscesses, but when the cavity is small there might not be space to accommodate sufficient tube to allow of the pus gaining access to the eyelets.

Translations.

BLACKWATER FEVER. (HÆMOGLOBINURIA.)

By R. KOCH.

Translated by P. FALCKE.

(Continued from page 137, December number, 1899.)

No. 9.—The patient has been in East Africa for fifteen months. Only three weeks after his arrival he had his first attack of malaria, and two months subsequently the first attack of blackwater fever, a few hours previous to which he had taken quinine. Since then, an attack of blackwater fever followed almost every time he took quinine. According to his statement he had ten such attacks, which much weakened him. When I, by chance, saw the patient, he was very anæmic and had daily attacks of fever ushered in with severe rigors. The doctor who was treating him once more gave quinine a trial, and gave him 1.0 grm. This was followed a few hours later by severe rigor and urine tinged with blood. The rise of temperature quickly subsided and next day the patient was already fever-free. The urine still retained its bloody colouring for a few days. The fever only continued five days and on the sixth day the relapse already set in. Then a trial was made with arsenic treatment, entirely without result. The patient daily had an attack of fever with temperature which ranged as high as 40° C. and over. At this time, for a few days, I examined the blood of the patient and found two generations of large, pigmented parasites; incidentally also the characteristic forms of sporulations. He thus was suffering *tertiana duplex* quite independently of his daily attacks of fever. Of the parasites of tropical fever there was never a trace. In this case it was a question of a quite pure tertian.

As the arsenic had proved unreliable, nothing remained to be done but to try quinine once more with the greatest care. For this purpose the patient was admitted into hospital, and it was resolved to give him only 0.5 grm. quinine, and that subcutaneously. The patient was previously observed for another day, and several blood examinations undertaken. Hitherto the attack of fever had always begun between midday and 1 p.m. On this day, also, the rigor set in at 12 p.m. Besides adult tertian parasites and sporulation forms, parasites of medium size were exhibited in the blood at the same time. Thus it was still a case of a *tertiana duplex*. The further examinations, also, undertaken on the same day, agreed with this condition. At 8 a.m. next morning 0.5 grm. quinine bimuriate was administered subcutaneously. At 10 a.m. (two hours earlier than the fever was wont to set in) the patient was attacked by an extraordinarily severe rigor, which lasted half-an-hour. Simultaneously pains in the limbs, vomiting, restlessness, extreme weakness and prescience of death set in. The colour of the skin changed visibly and became of a yellowish tint. Immediately after the rigor the patient voided 250 cc.m. of bloody, blackish-red urine. At about two o'clock 150 cc.m. of black-red urine was passed. From that time the weakness of the patient rapidly increased, he became somnolent,

and at 10 p.m., ten hours after the injection, death occurred.

Shortly before the injection of quinine, and also during the attack, the blood, the same as on the previous day, contained two generations of parasites. A little time before death there was only one generation, the other generation had disappeared in consequence of the effect of the quinine. At the *post-mortem*—with the exception of exceptionally great swelling of the spleen and icteric discolouration of all the organs—there were no changes.

In this case the endothelia of the spleen and liver contained a great deal of blackish pigment.

From the foregoing statements it will be gathered:

(1) That the malaria parasites are very often lacking in blackwater fever.

(2) That when they are present their number stand in no relation to the hæmoglobinuria, as should be the case according to the analogy of the Texas fever.

(3) That there is malaria with very numerous parasites without hæmoglobinuria developing therefrom.

(4) That on more narrow investigation essential clinical differences are revealed between the attack of malaria and that of blackwater fever.

(5) That blackwater fever may be combined with two quite different types of malaria, namely with the ordinary tertian and with the tropical fever.

By these results I consider ample proof is afforded to prove that blackwater fever is not malaria, but a disease which can set in independently, but may be combined with malaria more or less frequently.

Notices of Books.

WE are glad to know that a "Handbook on Gnats or Mosquitoes,"—the *Culicida*—by Major George M. Giles, I.M.S., M.B.Lond., F.R.C.S., is in the press. A work of the kind is much wanted at the present moment, and the fact that so eminent an authority as Major Giles is the author guarantees its value. The attention of those intending to obtain the Handbook should be drawn to the conditions. Subscribers paying in advance are charged 10s. 6d.; when purchased after publication the book will cost 15s. net. As every medical practitioner resident in the Tropics will be bound to possess a copy of this work, an early application is desirable, before the edition is exhausted. Messrs. Bale, Sons, and Danielsson, Ltd., 85, Great Titchfield Street, London, W., are the publishers.

New Foods, Drugs, &c.

PLASMON.—It would seem as though a new, valuable and permanent addition had been made to our stock of invalid foods by the introduction of Plasmon. Experience alone can determine whether this food will come up to the expectations of its introducers in all points, but that it has already fulfilled many, if not most of the virtues claimed, seems an established

fact. Without dealing, however, with generalities, it is within our knowledge from personal trial that Plasmon, in tropical intestinal ailments, is of the highest value. At once nutritious, easy of digestion, and pleasant to take, Plasmon fulfils what so many medical practitioners in the Tropics have been searching for, namely a nitrogenous food unhampered with accessories which render assimilation impossible, and which, by the excrementitious matter produced, teases the gut and irritates its bare or ulcerated surfaces. Milk is condemned by many as a food in tropical intestinal ailments; it is found that in many cases of sprue, chronic diarrhoea, and dysentery that milk is unsuitable, and it is laid aside and preference given to a "meat" diet; but "meat" diet has several objections in such diseases. Plasmon—a highly concentrated milk proteid—promises to provide a food which removes what is objectionable in a milk diet pure and simple; and, what is more to the point, a practical trial in these very diseases has been attended by most beneficial results. There is no doubt that practitioners in the Tropics will find Plasmon an essential food in the treatment of intestinal ailments, and it has only to be brought to their notice to at once establish its use and usefulness. The Company promoting Plasmon is composed of men whose names are an ample guarantee that the quality of the food will not deteriorate as it becomes more "fashionable"—a most necessary factor to note in the recommending of any specific food for invalid use.

The address of the Plasmon Syndicate is 56, Duke Street, Grosvenor Square, London, W.

News and Notes.

WE are sorry to record the death of Rudolph Plehn, D.Ph., who was born on February 23, 1868, and shot by natives in the bush near Bertna, South Adamana, on November 24, 1899. He made many political and scientific expeditions in Togo. He was the leader of the Lango-Ngoko expedition into Cameroon in 1898, and to him may be ascribed the opening out and cultivation of the south of this colony, which until then was almost unknown. He was the youngest brother of Dr. A. Plehn, the Imperial Governmental Physician of Germany, and of Dr. F. Plehn, both of whom have contributed largely to our knowledge of tropical diseases, especially in regard to Cameroon, where their energetic and talented brother met his untimely death.

A PLAGUE LABORATORY IN ST. PETERSBURG.—This laboratory, established by the Russian Government, for the study of plague and the preparation of anti-plague serum, is situated in a building within the fortress of Cronstadt, and completely isolated from the rest of the fortress by being surrounded by water. The staff and assistants are provided with apartments and the horses necessary for laboratory work are stabled on the island. The possibility of the conveyance of infection is reduced to a minimum. These elaborate precautions are in every way justified, whilst

the catastrophe which occurred at Vienna is borne in mind.

A NEW QUININE PREPARATION—BASICIN.—Under the name basicin a new preparation of quinine has been placed upon the market. Its composition is not disclosed, but it is certainly a combination into which the alkaloid caffeine enters. Its antipyretic value is claimed to be far higher than that of quinine itself.—*British and Colonial Druggist*.

ABSCESS OF THE LIVER.—Lucas Championniere states that by means of the radiograph the presence of a "tumour" in the liver was made evident. Subsequent exploration proved the "tumour" to be pus, which was successfully operated upon.

PREVENTIVE INOCULATION AGAINST PLAGUE, by a nucleo-proteid extracted from plague bacilli, has been notified by A. Lustig and G. Gallotti. The discoverers of this preparation claim that their product is free from the drawbacks of Haffkine's vaccine.

BILHARZIA HEMATOBIASIS IN U.S. AMERICA.—Dr. E. Walker reports a case of this disease as occurring in Sparta, Illinois, U.S.A. Great care was taken to avoid error in this diagnosis. The observation is important, as hitherto the disease has been reported only from Africa.—*Medical Record*, February 24, 1900.

A CURATIVE HUMAN SERUM FOR YELLOW FEVER.—Injections of serum, taken from persons convalescent from uncomplicated cases of yellow fever, have been tried by A. Agramonté, with apparent benefit. The human serum is more readily absorbed than the equine serum. The injection is never given later than the fourth day after invasion. The result has been that the disease has been distinctly modified in every instance. Two facts have been emphasised, namely, the absence of hæmorrhagic tendency and the rapid convalescence.—*Medical Record*, February 24, 1900.

PERSONAL INFECTION IN TYPHOID FEVER.—Dr. Alfred Hill, Birmingham (Report, Fourth Quarter, 1899), states that he is convinced that direct contact with the person or belongings of patients suffering from typhoid fever is one of the greatest factors in the spread of the disease. Amongst the most important precautions are the strict isolation of the patient, cleanliness of the nurse, and of the patient's body and bed clothing, and immediate disinfection of the stools.

Correspondence.

WE have received permission to publish this letter. Dr. J. C. Thomson is on the staff of the Colonial Medical Service, Hong Kong.—EDITORS.

DEAR MR. CANTLIE.—I regret that another engagement this afternoon prevented my attendance at the last of your course of lectures on the "Surgical Aspects of Disease in the Tropics."

The whole curriculum of study in the School of Tropical Medicine has more than realised the first impressions I

described to you soon after I came into residence here. I think it remarkable that the organisation is already so complete, and the thoroughly practical character of the work being done makes the course a most useful one. Great credit is due to the Committee under whose management the School has developed so rapidly, and it seems to me that it should be more widely known that thus far the lecturers have given their services gratuitously. There is an admirable opportunity for the combination of philanthropy, with the furtherance of personal interests, in the endowment of Lectureships, or the creation of a Lecture Fund in the Tropical School, by some of our wealthy merchants and others who have such extensive concerns in the tropics and sub-tropics, which are to reap the direct benefit of the increased skill in methods of diagnosis and investigation that the work of the School will soon place at the disposal of English-speaking people resident all over the world.

The School is fortunate in having such an able and enthusiastic scientist with practical experience of the tropics as Dr. Rees to conduct the laboratory work; and it conduces to very great thoroughness of study that each item on the Syllabus, as it is dealt with by the systematic lecturers, is at once revised and gone into from the practical points of view by Dr. Rees, in the laboratory.

Will you allow me to criticise the arrangements at one single point? If you agree with me, perhaps you may move in the matter in Committee. Each of the three sessions in the year consists, as you know, of an Eight Weeks' Lecture Course with practical work running concurrently with the systematic lectures, followed by another month's laboratory for those who are able to continue for the longer period. I think it will be found that most men—and women too, for we have had four lady students this session—will join for the Eight Weeks' Course. Now it has seemed to some of us unfair to men, who do enter for the full course, to permit new students to drop in all through the session, as is done at present. During the second month Dr. Rees has had new men commencing every day or two, and while he is most patient in doing his level best for every man who enters, it appears to me that there is a great and useless dissipation of energy in allowing things to go on in this way. Necessarily, where so many sets of beginners are being attended to, the time available for the senior students becomes less and less as the session advances. It would meet the case and obviate this definite weakness in the present scheme if men, who want to attend for a shorter term than eight weeks, were limited to joining either at the beginning of the session, when elementary work is being done and first principles inculcated, or at the close of the Eight Weeks' Lecture Course, when a new short practical course could be taken up by the tutor. This would leave the tutor free to spend the second month of each session in carrying the original set of students on more systematically to advanced work than is now possible.

I am booked to leave again for the far East early next month. I wish this course of study had preceded my first departure, now nearly twelve years ago.

Yours very truly,
JOHN C. THOMSON.

IDENTITY OF MAHAMARI AND PLAGUE IN CENTRAL ASIA.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—I am much interested in Dr. G. Clemow's paper on "Plague in Central Asia," as the outbreaks described are evidently identical with the *mahamari* of Gurhwal, a Himalayan district on the northern frontier of the N.W.P., India. It would be easy to unearth from the records of the Sanitary Commissioner's office almost exact duplicates of the reports he quotes, as regards the human victims of the disease, but no species of marmot is, at any rate, common in the in-

habited parts of the region, though it is likely enough that plenty may be met with in the more elevated parts of the mountains, as further to the west, on the Pamir, I met with enormous numbers of an *arctomys*. I forget the species to which I referred it, but it was not *bobac*.

In Gurhwal the animal affected was the common rat.

At the time I was called upon to investigate the history of certain outbreaks, plague had not appeared in India proper, and the soothing opinion prevailed that the malady was nothing more than typhus; but after visiting the villages that had been affected, I was convinced that the disease had little in common with typhus, but was undoubted Oriental plague, and indeed reported it to be such.

There exists a good deal of trading communication between Gurhwal and Central Asia, as one constantly meets with Mongolian caravans that come down to exchange wool and skins for salt and other commodities of which they stand in need, and it is obvious that if the disease be endemic in the conterminous parts of the central plateau, the disease might be easily introduced through the agency of infected skins and other merchandise, for the march is too long an one for it to be likely to be carried by infected human beings.

GEO. M. GILES, M.B., F.R.C.S.

Byfield, Mannamead,

Plymouth, February 17, 1900.

Major I.M.S.

Letters, Communications, &c., have been received from:—

B.—Major W. J. Buchanan (Bhagalpur); Major E. H. Brown, L.D.S. (Calcutta).

M.—Insp.-Gen. Edw. Mahon (Haslar); Dr. W. A. Mackay (Huelva).

P.—Dr. Fredk. Pearse (Calcutta).

S.—Dr. Ad. Sechehaye (Geneva).

W.—Dr. Percy d'Erf Wheeler (Jerusalem).

Y.—Major Yarr, R.A.M.C. (Woolwich).

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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

TICKS AND TICK FEVERS.

By L. W. SAMBON, M.D. (Naples).

Lecturer to the London School of Tropical Medicine.

THE popular belief that the bite of ticks is venomous is very old and widely distributed, but naturalists have hitherto disregarded it because of the absence of special poison-glands in ticks, and because of the immunity of numberless hunters, field-naturalists, and farmers repeatedly attacked by these pests.

That ticks are not poisonous animals in the same way as certain snakes and insects is undoubtedly true, but, on the other hand, there is ample and reliable evidence to prove that their bite may be occasionally followed by serious illness. To explain this fact, several authors have suggested the possible entrance of pathogenic micro-organisms from without into the seat of puncture, and this may be true in a proportion of cases, but recent discoveries concerning the direct transmission of pathogenic parasites by means of intermediate animal-hosts has thrown a new light upon the rôle played by ticks in the causation of certain diseases.

In this article we shall summarise very briefly the little that is positively known on the subject, in the hope that it may lead to further investigation.

The ticks, or wood-lice as they are commonly called, are allied to the spiders. They belong to the order of the *Acari* of which they are by far the largest specimens. They all lead a semi-parasitic life on land vertebrates, and although the various species have their peculiar zoological distribution,

yet most of them may be found accidentally on a variety of unusual hosts. Most species have a restricted geographical distribution, but a few that are parasitic on migratory birds or on widely distributed domesticated animals are almost cosmopolitan.

The life history of the ticks is pretty well known in its broad lines:

The newly-hatched ticks are called *larvæ*. When they emerge from their egg-shells, they are nearly white, but they soon acquire their peculiar colour and resemble minute seeds. These *larvæ* possess only six legs and, apparently, no sexual organs. According to Curtice, they seem to breathe through three stigma-like dots placed just behind each pair of limbs. The *larvæ* soon climb the stems of the nearest shrubs, and clasping the tips of the leaves with their two posterior pairs of legs, await, with the front pair outstretched, ready to cling to any animal that may sweep through the herbage. Having found a suitable host, they fasten themselves by inserting their *rostrum* or beak into the skin of the animal, and remain thus adherent for two or more days, to obtain nourishment. Having become sufficiently gorged, they let themselves fall to the ground and remain concealed for some time in order to undergo a first metamorphosis.

This metamorphosis consists in the addition of a pair of legs behind the others and of a pair of large stigmata behind them. The tick, in this stage of life, is called a *pupa*. After the metamorphosis, it climbs the stalks of herbage just as it did in its larval stage, and again awaits patiently the passage of a host. After a few days, the *pupa*, having been sufficiently nourished, drops once more to the ground to undergo its second and final change.

This second metamorphosis leads to the adult stage

with perfectly developed reproductive organs. On reaching the adult stage, both males and females again await on herbage for a passing host. During this stage, sexual intercourse takes place. It is a remarkable proceeding, during which the rostrum and other mouth organs of the male are inserted into the orifice of the sexual apparatus of the female, which is situated between the base of the posterior pair of legs. After copulation, the male tick dies. The fertilised female, having become enormously distended through blood-suction, drops off to lay her eggs in some safe and convenient place. The number of eggs varies with the species, but it is always enormous. Mégnin counted over 12,000 eggs laid by the common cattle tick, and Barber as many as 20,000.

The eggs are laid one by one, and each one is in turn coated with a glutinous protective substance which is secreted by a pair of racemous glands situated just under and within the head-shield. The object of coating the egg has been clearly demonstrated by Berskau, who found that eggs laid after destroying the coating sac and preventing the eggs being covered dried up and would not hatch, while others previously laid by the same female and coated hatched in due time. Oviposition lasts about a week, and while the pile of eggs grows larger the body of the animal contracts, until nothing more is left than a yellowish, dried up, shrivelled skin, whence all life has departed.

The eggs consist of a thin, shell-like covering with a dark, opaque mass within. In the latter stages of incubation the form of the larval ticks becomes more and more apparent. The enormous quantity of blood sucked by the female ticks serves as a food supply for its progeny; in fact, it becomes the vitellus of which the eggs are largely supplied, and residues of which may be seen within the abdomen of the young larvae after birth.

The life-span of ticks varies greatly in accordance with surrounding conditions. It may last only one season or be extended through several years. Warmth and a certain amount of moisture are favourable conditions to rapid development, but the most essential is the immediate insurance of a suitable host at the completion of each metamorphosis. No further development seems possible without a new supply of food.

To meet the uncertainty of finding at once a suitable host, nature has endowed ticks with extraordinary powers of fasting. Between each metamorphosis they may remain several months without food. Some specimens of a Persian tick (*Argas persicus*) sent to Dr. Mégnin were still alive after four years' fasting. Similar instances of long-enduring resistance to want of food in amphibia, mollusca, and insects are well known to naturalists. A curious instance is that of some land-snails in the collection of the British Museum. They had been kept for years with their shells glued down, but one day, under specially favourable meteorological conditions, they calmly crept away from their labels.

The ticks are very numerous in species. Zoologists have grouped them in one large family, *Ixodes* which is sub-divided into two sub-families, *Ixodinae* and *Argasinae*. The *Ixodinae* are further divided into several genera and the *Argasinae* into the two genera, *Argas* and *Ornithodoros*.

The *Argasinae* are easily distinguished from the *Ixodinae* by their coarsely granular skin, flattish bodies, and the entire concealment of the head-like process (*capitulum*) beneath the projecting for-margin of the cephalothorax. The *Ixodinae* have a carpace or shield which covers almost entirely the body of the males, but is very small in the females, being limited to the anterior part of the cephalothorax so that it may not interfere with the distention of their bodies.

Throughout life the male tick enlarges but little; the female, on the contrary, grows to ten or twenty times its size, and her body becomes quite globular, so that she can scarcely be recognised as of the same species as the males.

All ticks are provided with a *rostrum* or beak formed by the two mandibles above and the labium below. The labium is armed with recurved barbs. The sucking apparatus lies in the mandibles, up each of which run two tubes designed for this office. By means of this beak, which is inserted into a follicle of the skin, the tick adheres firmly to its host. The distended females are kept in position solely by the inserted rostrum, their legs having become useless on account of the abdominal swelling.

The injury inflicted by the ticks is hardly noticed at first, but the sensation becomes troublesome as the animal becomes gorged; there is a certain amount of irritation around the point of insertion of the rostrum, forming a transitory wheal. This is probably due to the irritating nature of their salivary secretion, which is purposely injected to draw blood to the spot, as is the case with all non-poisonous blood-sucking insects. As the tick withdraws its beak a minute drop of blood follows, filling the pore and showing externally as a very small dark spec. If the attempt is made to remove the tick forcibly, the rostrum is frequently broken off and remains imbedded in the skin. When this occurs, a slight leucocytic process follows and eliminates the rostrum just as if it were a thorn.

Some species of ticks, such as the "sheep-tick" (*Ixodes redwivius*) may occasionally penetrate entirely beneath the skin after the fashion of other acari, of which the itch-mite (*Sarcoptes scabiei*) is a well-known type. But, even then, they never give rise themselves to any but trifling local symptoms. In 1891, Professor R. Blanchard described such a case at a meeting of the *Société de Biologie*. A middle-aged man came to hospital for a disease of the articulations. While being examined, he said he had noticed for some weeks past a small swelling on his belly, which gave him no trouble whatever, but of which he desired to know the nature. He presented, in fact, on the right side of the abdomen, at about the height of the navel, a kind of subcutaneous tumour which was movable and looked very much like a sebaceous cyst; the skin offered no trace of inflammation, nor the cicatrice of any lesion through which a foreign body might have been introduced. The tumour was then lanced, and it was found to contain a living female tick (*Ixodes ricinus*), which had lodged itself in the subcutaneous connective tissue. Other similar cases have been reported by Beauregard, Choupe, Demos, Mégnin and others.

In medical literature, there are several cases

recorded of abscess, œdema, lymphangitis, adenitis, urticaria, tetanus, anthrax and glanders attributed to the bites of various species of ticks. These cases are of little importance to us, because, even if it were undoubtedly proved that the different diseases enumerated were in each case directly inoculated by ticks and not caused by a subsequent infection, their transmission must have been purely accidental. The diseases we propose to consider here are those which are constantly and necessarily carried by ticks from one to another of their ordinary hosts. Amongst such are the Red-water fever of cattle, transmitted by the common cattle tick (*Rhipicephalus sanguineus*), and one or more fevers inoculated to man by certain species of Argas.

Rhipicephalus sanguineus and Redwater Fever.

The old popular belief that cattle-ticks were the cause of redwater fever, or Texas fever, was lightly thought of, until the experimental researches made by Smith and Kilborne demonstrated that these acari were certainly concerned in the etiology of the disease.

Smith and Kilborne discovered that red-water fever was due to an hematozoal parasite belonging to that group of protozoa which causes malarial fevers in man. They named it *Pyrosoma bigeminum* because of the flame-like shape it acquires in certain stages of development and because two parasites are frequently found close together in the same hematocyte. *Pyrosoma bigeminum* does not offer the multiple, rosette-like segmentation which characterises the parasites of tertian, quartan and summer-autumn fever, but reproduces itself by simple binary division. I believe I was the first to point out this fact, which was later demonstrated by Laveran and Nicolle.

Smith and Kilborne proved also that ticks were absolutely necessary in the transmission of redwater fever, because in their experiments healthy cattle did not contract the disease when enclosed together with infected animals which had been entirely freed from ticks, whilst they acquired the disease without coming in contact with infected cattle, provided they were allowed to graze in fields infested by ticks which had dropped off infected animals. At first it was thought that cattle contracted the disease by ingesting the ticks together with their fodder; but this was disproved by the negative result of feeding experiments with adult ticks, tick eggs and grass from infected fields. It was also ascertained that the excreta of infected cattle do not communicate the disease.

Thus we gather that the rôle of ticks in the transmission of redwater fever is not a mere accident, but that they act constantly and necessarily as intermediate hosts to *Pyrosoma bigeminum*, just as certain gnats subserve in the life cycle of the malarial parasites of man. Without ticks cattle cannot become infected with redwater fever, just as man cannot become infected with malarial fevers unless the special parasites be inoculated into his blood by mosquitoes. This fact may seem doubtful to those who are not conversant with natural science, but it is strictly in accordance with the laws of parasitism, and many analogous examples might be adduced. The best known are filarial infection (*Filaria nocturna*), which is inoculated by gnats, and "*Nagana*," an African cattle disease,

which is transmitted by the tsetse fly (*Glossina morsitans*).

The most interesting feature in the experiments made by Smith and Kilborne is the demonstration that redwater fever may be transmitted by young ticks hatched in the laboratory, and that therefore the parasite must pass from the mother tick to her eggs. This fact was at first generally discredited, but Koch repeated Smith's experiment and confirmed it. However romantic it may seem, this fact is by no means unique; *Pebrine*, a protozoan disease of silkworms, is likewise transmitted, through the eggs, from the moth to the young caterpillars.

As yet we know nothing of the life history of *Pyrosoma bigeminum* beyond the little that has been observed in the blood-examination of infected cattle. The parasite has not been demonstrated in the tick, within which it probably undergoes a series of metamorphoses corresponding to those of our malarial protozoa. From our present knowledge of the life-history of ticks it is reasonable to infer that redwater fever may be transmitted by these acari both in their pupal and adult stages. Experiment has positively proved that they may transmit it in their larval stage. This, we think, may be explained by supposing that the parasites pass to the eggs with the vitellus which is derived from the infected blood imbibed by the fertilized female tick.

The *intra ovum* inclusion of parasites is by no means uncommon; a trematode, *Distomum ovatum*, may be found occasionally in the white of the eggs of fowls, having been enveloped in the albumen during its excursions into the oviduct. Nematodes have likewise been found enclosed in hens' eggs.

The cattle tick which is concerned in the transmission of redwater fever has received various names at different times and in different places; thus it has been called *Ixodes bovis* by Riley in 1869, and *Boophilus bovis*, by Curtice in 1890. These authors thought they were describing a tick peculiar to American soil. But *Boophilus bovis* is no other than the tick called *Ixodes sanguineus* by Latreille in 1806; *Ixodes plumbeus* by Dugès in 1834; *Ixodes dugesi* by Gervais in 1844, and *Rhipicephalus sanguineus* by Koch in 1847. It is the same tick in Europe, in Egypt, in America, and was everywhere introduced with imported cattle. Curtice thinks that it was introduced early in the sixteenth century into the Spanish settlements of America, and thence spread with the cattle to all such places as offered suitable surrounding conditions. Its original habitat was probably in the North of Africa. Curtice further expresses the probability that redwater fever, which was also called Spanish cattle fever, may have been introduced at the same time from the old world with the cattle and their ticks.

Redwater fever is not limited to oxen; sheep, and occasionally horses, also suffer from hæmoglobinuric fever. Man likewise is attacked by a disease indistinguishable from redwater fever in clinical features and *post-mortem* findings, but authors are not agreed as to its etiology. The hæmoglobinuric fever of man, generally called blackwater fever, is a disease of great importance on account of its deadliness and wide geographical distribution; it is to be hoped, therefore, that its causative agent may soon be definitely esta-

blished. Unfortunately, there are certain theories concerning its etiology which hitherto have greatly hampered judicious investigation. One of these is the quinine theory propounded by Tomaselli and lately supported by Koch, but the most popular is the one which considers blackwater fever to be nothing more nor less than an aggravated paroxysm of the ordinary fever in malarial cachectics. This theory has no support beyond the fact that the parasites of the ordinary malarial fevers have been found pretty frequently in the blood of patients suffering from blackwater fever. Nothing could be more unsatisfactory than this theory. In fact, in by far the largest number of cases no parasites could be demonstrated (Manson, Daniels, Koch, Sambon). In cases in which parasites of summer-autumn fever were known to exist, they entirely disappeared during the blackwater fever attack. In a few cases the hæmatozoa of tertian fever were observed (Manson, Hughes, Rees, Sambon). In others the early forms or the crescentic forms of summer-autumn infection (Manson, Plehn, Koch, Thin, Rees, Sambon and others). Admitting for a moment a connection between these microscopic findings and blackwater fever, the question would arise: which of these different species of parasites is responsible for the hæmoglobinuric fever? Both *Hamamæba vivax* and the hæmatozoa of summer-autumn fever give rise each to a peculiar type of fever which is constantly the same, varying only in intensity, but the course, duration and symptoms of blackwater fever are very different from those of either tertian or summer-autumn fever.

The fact that the parasites of tertian and summer-autumn fever have been found in the blood of blackwater fever patients does not prove that they are the cause of the disease. We must remember that in many places the ordinary malarial fevers and blackwater fever are co-endemic; we should therefore expect to meet with mixed infections of blackwater fever and tertian, or blackwater fever and æstivo-autumnal fever, just as frequently as with mixed infections of tertian and æstivo-autumnal, or tertian and quartan.

But further discussion would be useless, because the theory which ascribes hæmoglobinuric fever to ordinary malarial infection is based on two false premises: 1st, that it is an aggravated attack of ordinary malaria, and 2nd, that it appears only in malarial cachectics.

To say that a paroxysm of blackwater fever is an unusually severe manifestation of tertian or summer-autumn fever is not only false, but absurd, because attacks of blackwater fever may be exceedingly mild, whilst on the other hand the most severe and deadly infections with ordinary malarial parasites never produce hæmoglobinuria in regions in which blackwater fever is not endemic. As to the old belief that blackwater fever is witnessed only in malarial cachectics, we need but refer to recent literature on the subject, which reports many unmistakable examples of early attacks in new arrivals within the endemic regions of the disease, notwithstanding the absence of a previous malarial infection.

In studying diseases, we should not forget that much information concerning their etiology may be gathered from their geographical distribution and

epidemiology. This is certainly the case in blackwater fever, because neither its distribution nor its epidemiology do coincide with the distribution and epidemiology of the ordinary malarial diseases.

In previous papers I have expressed the opinion that the causative agent of blackwater fever must be specifically different from those of ordinary malarial fevers and that it is probably analogous, if not identical with that which causes hæmoglobinuric fever in cattle. However this may be, I think it would be well to investigate whether ticks have anything to do with the transmission of blackwater fever to man.

The west coast of Africa was considered at one time to be an endemic centre of yellow fever, but the experience of recent years seems to suggest that the yellow fever of the old authors must have been in reality blackwater fever. If this be so, the following observations indicate a possible connection between ticks and blackwater fever.

Dr. Alexander Bryson, in writing fifty years ago about yellow fever (blackwater fever?) in Bathurst, W. Africa, says: "It then took an eccentric course in the town, but prevailed principally in Water Street. It raged epidemically in August, and terminated in September. The question may be asked why it should have raged in Water Street, and answered by stating that it was in that street the European merchants chiefly resided. . . . There is another circumstance connected with the above street, which it may not be irrelevant to mention. Vast quantities of untanned hides are brought down the Gambia in small vessels, which not unfrequently arrive in a wet and semi-putrid state; consequently, when they fall into the hands of the merchants, they cause them to be spread out in the front of the houses and along the left bank of the river, to be dried by the sun, whence there arises an effluvium which could only be tolerated by those who are profitably engaged in the trade. Whether this had any effect in causing the disease to infest Water Street, I shall not hazard an opinion."

Dr. Horton, in his book on the Climate of the West Coast of Africa (p. 238), says: "The first case of yellow fever was in a large French house. The clerk, who was very temperate, as most Frenchmen are in tropical climates, was attacked under circumstances of a rather peculiar nature. He was up in the morning in perfect health, and was engaged weighing some cow-hides. There was among them one hide which had not been properly dried, and had become putrefied, and the smell from it made him vomit a great deal. Even the native labourers who were employed were sick from it. The clerk took to his bed, black vomit came on, and he died from a decided and rapid case of yellow fever."

Ticks, like fleas and lice, very soon leave the body of a dead host and drop to the ground, but when cattle are skinned immediately after death a number of ticks may remain attached and be packed and forwarded with the hides.

Conti and Silvestrini, who studied blackwater fever in Sardinia, mention the occupations of their patients, and thus we know that those attacked were mostly cattle herds and farmers.

Ticks abound in all the regions in which blackwater

fever prevails. In some districts they are especially numerous, and men have to wear thick boots and wrap their legs in puttees, not to be harassed by these abominable pests.

The prevalence of ticks varies greatly in different years. Some years they literally swarm, just like locusts. It would be interesting to know whether there be any connection between their prevalence and the prevalence of blackwater fever, which is also known to be very frequent in certain years and almost absent in others.

Ixodes reduvius and Louping Ill.

Professor Williams published in 1897 an account of certain experiments made by him, which seem to prove that louping ill may be conveyed to sheep by their ticks. Four sheep were muzzled, and then turned out on infected ground at Leithen Hall farm, Edinburgh. The only food they were allowed to eat was given them indoors. They were soon attacked by ticks, and, of the four, two died of louping ill. A fifth, a ewe, was kept indoors. Some ticks were placed on her, and ten days after she contracted the disease and died, without having been near the affected farm. Of other four sheep that he inoculated with cultivation material taken from the spinal canal of a sheep that died of louping ill, three contracted the disease and died.

In the same year Messrs. Meek and Greig-Smith published a paper stating that they had succeeded in cultivating a bacterium taken from wounds caused by ticks on sheep. Two rabbits, on being inoculated with this, exhibited all the symptoms of louping ill.

The tick which is concerned in this disease is *Ixodes reduvius* (de Geer), an acarus exceedingly common among sheep in Europe. When it attacks sheep in its larval or pupal stages it is found principally on their faces, and is therefore popularly known as "face tick."

Ixodes reduvius is found also on goats, oxen, horses and dogs; it frequently attacks men, and several cases have been reported of abscess, ulceration and fever attributed to its bite. In its larval stage it attacks a large variety of mammals, birds and reptiles. Mégnin found it on lizards, greenfinches, gulls, bats, ferrets, hedgehogs, hares, squirrels and deer. A careful study of the zoological distribution of this tick may possibly help to elucidate the etiology of louping ill. We know that the disease is caused by a bacillus, but nothing is known of the life history of this micro-organism, which may or may not be the parasite of one of the many hosts of *Ixodes reduvius*.

Argas persicus and the Disease of Miana.

Several travellers have reported on a peculiar disease of Miana in the north of Persia, which the natives unanimously attribute to the bite of a tick (*Argas persicus*, Fischer). According to Kotzebue and Waldheim, the bite of this argas is followed by severe pain, delirium, convulsions and sometimes even death. Schlimmer says the symptoms are like those of "remittent fever—extreme lassitude, disinclination to work, yawning, fever, perspiration, not accompanied by much thirst, increasing and decreasing at stated hours in the day." Fatigued travellers, and those who have undergone privations are especially liable. Schlimmer relates

that he once treated 400 soldiers who claimed to have been bitten by these parasites at Miana. Taschenberg believes that the effects ascribed to *Argas persicus* are really due to a fever which prevails in Miana. Dr. Tholozan, who at first did not believe that the bite of the argas could produce disease, completely changed opinion after a long residence in Persia, and concludes from a number of observations "that it is no longer possible to doubt the danger from the bite of these animals, and that the number of accidents is very great." Natives are comparatively immune, and Schlimmer considers that this immunity is acquired by their having been bitten at some time or other by the tick. According to Dupré the natives treat the disease by prohibiting the use of meats and of acid or fermented beverages, and by administering sugar which they consider to be a specific.

The *Argas persicus* has a flat, thin body of a clear blood-red colour, spotted on the back with a great many white granulations. Its legs are pale yellow. It is found in the north and east of Persia, and is called *guérib-guez malleh* at Miana, and *keuch bhebquez* at Chahronde and Bestham, on the main road from Teheran to Khoragan. Its habits are similar to those of the common bed-bug; it infests old houses, living in the cracks of walls and floors. Kotzebue says that it may so infest villages as to drive out the inhabitants.

Professor Laboulbène received a number of *Argas persicus* from Dr. Tholosan, and Mégnin allowed himself to be bitten by one of these. The bite was not followed by any kind of illness. From this experiment, and from others made on rabbits, the two French authors conclude that the bite of *Argas persicus* is not dangerous. This statement is quite correct, but Mégnin's experiment does not prove that disease may not be transmitted through the bite of this tick, because Mégnin's argas had not received food for four years. Then again, it is not likely that every individual argas is capable of transmitting the Miana disease, any more than every individual anopheles is capable of transmitting malarial fever. To be capable of infecting a person with tertian fever or summer-autumn fever, the mosquito must have first become infected itself by imbibing the blood of a patient suffering from tertian fever or æstivo-autumnal fever.

Ornithodoros Moubata and the Disease of Tete.

It was Dr. Livingstone who first described the tick disease of Portuguese South Africa. He met with it at Ambaca and at Tete on the Zambesi river. He says: "The effects of the bite are a tingling sensation of mingled pain and itching, which gradually ascends the limb until it reaches the abdomen, where it soon causes violent vomiting and purging. When these effects do not follow, as we found afterwards at Tete, fever sets in; and I was assured by intelligent Portuguese there that death has sometimes resulted from this fever."

Several years later Sir John Kirk again mentioned this disease, which he also found in the Zambesi valley as far up as Sescheke, above the Victoria Falls. He writes: "The symptoms appear soon after the bite, and are sharp fever, vomiting and often delirium; in about two days these pass off, but there is no marked

profuse perspiration as in malarial fever. After recovery, the patient has complete immunity from further attacks, however he may be bitten, but it is doubtful whether this immunity lasts for any length of time in case of removal."

Owing to the kindness of Mr. Pocock and Dr. Manson, I am able to add some further information on the subject.

Mr. Pocock showed me a letter by Dr. Dowson of the Royal Navy, who presented the Natural History Museum with several specimens of the Tete tick. In his letter Dr. Dowson says, "The Portuguese believe that the severity of the subsequent symptoms depends on the number of bites received. Severe fever and dysentery, with great swelling round the anus, are stated to have occurred. . . . A Dutch trader assured me that he was bitten by one of these acari on the toe, and that the bite was very painful. A few hours later he was seized with most violent abdominal pain, vomiting and purging. The stools contained blood. He was ill for three days afterwards, but said there was no fever at all."

Dr. Daniels, in a most interesting and valuable letter to Dr. Manson describing the work he is so ably carrying out in British Central Africa says: "I have come across the 'tick disease' Sir John Kirk told us about. It is only found for a very limited area on the west coast of Lake Nyassa and inland from that. All the natives agree as to the disease. Usually several ticks must bite, they produce a small lump which remains for two or three days. In from five to ten days they get an attack of abdominal pain with vomiting and purging. The stools are sometimes dysenteric. This lasts for nearly a week and is accompanied by fever and rigors. After that, fever continues often for two or three weeks. Some people are not susceptible, others get only one attack, and a few may have several, but in time they also become immune. The case I saw, an adult, was in the second week, and the purging had stopped. The temperature ranged from 100° to 100·8° F. Three blood examinations were negative. There were no physical signs."

The tick which gives rise to this dysenteric disease in Tete is called *Kufu* or *Bu*, by the natives and *Tampan* or *Garrapato* by the Portuguese.

Mr. Pocock, the distinguished arachnologist of the British Museum, considers the Tete tick to be identical with *Ornithodoros moubata* found by Dr. Welwitsch in Angola, and described by Mr. Andrew Murray under the name of *Argas moubata*, after the local native name.

Ornithodoros moubata has been confounded with *O. savignyi*, which occurs in Egypt and is common in Somaliland and British East Africa, but it differs from this species in having no distinct eye-spots, smaller processes on the legs and the dorsal surface of the body less pitted. The body is flattened from above downwards and is oval in outline. The prevailing colour of alcohol-preserved specimens is a dark greyish or brownish black with the extremities of the legs yellow. The integument is hard, leathery, covered with close set shining granules or tubercles and marked both above and below with symmetrically arranged grooves.

The geographical distribution of *Ornithodoros moubata* extends right across Africa along the Zambesi and its tributaries, and, as far as our present knowledge goes, seems confined to this river system.

In habits, *Ornithodoros moubata* resembles our common bed-bug; it inhabits old houses, hiding the day in cracks in the walls and floors and moving about actively during the night in search of nourishment. It attacks both man and beast.

The natives protect themselves in some parts from this pest by plastering their huts, both floor and walls, with mud and cow-dung. This practice is adopted by the Boers in their farm houses, and the Bechuanas and nearly all the cow-keeping tribes. The huts are also frequently smoked in order to drive the ticks from the thatch. The Portuguese at Tete have a great dread of this parasite and they always warn new arrivals not to place their beds on the ground, and to have the inside of their mosquito nets searched for this tick before retiring.

The Garrapatos of America.

In Central and South America, as well as in the West Indies, there are various species of ticks which are exceedingly troublesome to man and animals. Some of these seem to be carriers of disease germs, just like the ticks that we have already mentioned, but information is very scanty and unreliable. It is hoped that these ticks may receive the attention of students, so that we may become acquainted with their habits and the relationship they have to certain infections.

Argas talaje, Guérin-Méneville, occurs in Central America and Mexico; its rostrum may be entirely retracted. M. Sallé, the well-known French entomologist, who collected largely in Central America, thus describes the bite of this tick.

"Being at Casa Vieja de Gastoya, on the road from Guatemala to Zacapa (Central America), about fifteen leagues from Guatemala, May 6, 1847, I was awakened several times out of a profound sleep by atrocious itching on the hands and face, and my companion, M. Jules, suffered still more than I did. At three o'clock, irritated by these painful bites, I lighted a candle, and found that I had my hands covered with blood and blotches like large bites of bugs, which I supposed must belong to some particular species of monstrous size. My companion told me that we had been stung by wasps lodged in the walls of the house. On arousing the muleteer who conducted us, he told us that we were the victims of an animal called *talaje*, which they considered a large flea. On seeking for the insect I found this *argas*, which appeared to me very disgusting. Some were distended with blood, others had the skin rugose and wrinkled. I then remembered having taken some of them off my face during the night, and having rolled them between my fingers, taking them for some of the ticks with which my mule was covered to such a degree that some people told me it would be killed by them.

"These *talajes* keep themselves in the crevices of the walls of old houses. These walls are made of bamboos, roughed with mortar. They bite like fleas, and return to their holes before morning, for they are nocturnal. My hands and ears were much swollen,

and I suffered horribly. On piercing some of the pustules full of blood occasioned by these, a drop of blood issued. I washed myself and put in the water some drops of ammonia; but in place of allaying the pain, that only added to the swelling and inflammation. M. Jules did nothing, and suffered as much as I did, only the swelling was less and did not last so long." M. Sallé passed two bad, feverish nights after this. On the third he began to get better, but it was not until a fortnight afterwards that he was entirely cured.

Another troublesome tick in Mexico is the *Argas turicata*, A. Dugès, which is almost square in shape. Its usual host is the pig, but it also attacks man and its bite may be followed by serious illness. Pigs sometimes die from the consequences of its bite and chickens fed on these ticks die about the third day. Dugès reports three cases in man in which general symptoms followed the bite of this argas. In two of these a vein had been punctured by a turicata. One patient had difficulty in speaking and swallowing, swelling and numbness spreading over the whole body, vomiting and diarrhoea. In another patient all these symptoms subsided within an hour, when an urticaria made its appearance, accompanied by profuse perspiration. Dr. Aleman says some persons are refractory to the bite of the turicata, in others it usually gives rise to rigors, fever, headache and backache, which may last for several days; if the patient scratches the seat of puncture an ulceration may form and last for months.

Ticks are called *Nigna* in Carthagena, *Pique* in Peru, and *Garrapatos* in Mexico.

Preventive Measures.

Several measures have been suggested for the destruction of ticks, but, for the most part, they have failed to do any good. Some are totally inadequate, others cannot be put into practice in an efficient manner. It has been proposed to flood or fire infected land, but flooding is not possible everywhere and fire is not always controllable. Besides, a number of small mammals and reptiles escape to return as soon as the danger is over with the ticks they harbour. Since the ticks get access to their hosts mainly by being brushed upon them from the leaves of bushy plants or trees, it has been recommended to keep away the cattle from wooded pastures and to destroy all rank vegetation, bracken and rushes within their enclosures. For direct treatment there is probably nothing better than the dipping process, by which the animal is completely drenched with cotton-seed oil or some other kind of innocuous dip. Of course the dip only kills those ticks that are on the animal at the time of application and does not prevent further attacks.

For houses infested by *Argas*, nothing is more effectual than thorough fumigation with sulphur, or perhaps bisulphide of carbon. When this is not possible, all cracks and crevices should be sprayed with boiling water, kerosine or benzine. Then the walls should be white-washed taking care to plaster up all crevices.

A most valuable remedy for immediate use in a sleeping room is the powder of the pyrethrum flowers, which should be dusted between the sheets

of the bed. Some protection may be obtained by keeping a lamp alight by the bedside throughout the night.

When a tick has fixed itself on the skin, the only indication is to induce the voluntary detachment of the animal. This may be obtained by the application of a drop of olive oil, turpentine, benzine or petroleum on the parasite. The best plan is to rub its ventral surface with a feather or spill of paper dipped in oil, so as to choke its respiratory apparatus which opens on the belly.

Endeavours to detach the tick forcibly give rise to great pain, and the rostrum breaks off very easily, remaining embedded in the skin. In most places where the attack of ticks is of common occurrence great stress is laid in not tearing off the head of a tick that has implanted itself in the skin. Speaking of *Argas turicata*, Dugès says that the effect of the bite is especially bad if the rostrum be torn off, and where this occurs he recommends the use of the cautery.

THE ENDEMIC CENTRES OF PLAGUE.

By FRANK G. CLEMON, M.D., D.P.H.

II.

PLAGUE IN KUMAON AND GARHWAL.

THE story of plague in the endemic centres of the disease in China, which was briefly recounted in last month's JOURNAL OF TROPICAL MEDICINE, had not, I believe, been told completely or in a connected manner before. The case is different in regard to the history of the disease in its endemic home in India. The story of its behaviour there, from its first appearance until the beginning of the last decade, has been frequently told, and it will be unnecessary here to repeat in detail what is already familiar to most students of the disease. The literature of the subject is somewhat extensive, but an admirable summary of it will be found in a paper read by Colonel Hutcheson, I.M.S., before the First Indian Medical Congress in 1894.¹ It will suffice to relate briefly the main facts connected with the history of the disease before passing on to consider its behaviour within more recent years.

Kumaon and Garhwal, the scene of successive outbreaks of plague, lie approximately between the 28th and 31st degrees of north latitude, and between the 76th and 81st degrees of east longitude (from Greenwich). Garhwal is bounded on the north by Tibet, on the east by Kumaon, on the south by Bijnor, and on the west by Tehri and Dehra Dun. Kumaon is bounded on the north also by Tibet, on the east by Nepal, on the west by Garhwal and on the south by the Tarai, or malarious forest belt at the foot of the Himalayas. The two districts adjoin one another, and contain together a population of about one million souls, with an average of only seventy-two to seventy-six persons to each square

¹ "Mahamari, or the Plague, in British Garhwal and Kumaon." By G. Hutcheson, M.D., Brigade-Surgeon Lieut.-Col., I.M.S. *Transactions of the First Indian Medical Congress*, 1894.

mile. The large majority of the people are Hindus. Both districts occupy the lower southern slopes of the Himalayas. "The greater proportion of the population live at from 3,000 to 6,000 feet above sea-level, overlooked by the crowning peaks, rising from 20,000 to over 25,000 feet, of the great Himalayan range." The villages are scattered among the hills and valleys, and some of them cling to the mountain side in spots so difficult of access that they can only be reached "by means of a cradle, swung on a rope above a boiling flood of snow water."

From time to time outbreaks of a disease, locally called *mahamari*, occur in these districts, and from the description of the clinical characters of many of the outbreaks there is scarcely room for doubt that they have been epidemics of plague. The term *mahamari* appears to be used locally to indicate any severe disease, and it is just possible that some of the outbreaks called by that name have been of some other disorder, such as cholera. Some uncertainty seems to surround the application both of this term and of the term *sunjar*, which is also in use among the Himalayan hill tribes. *Sunjar* has been generally believed to indicate typhus, but an epidemic of a disease called by this name was investigated last year by Dr. Leonard Rogers, and proved to be one of relapsing fever; and it appeared from enquiries made on the spot that mild outbreaks of epidemic disease in these hill tracts are termed *sunjar* and severe ones *mahamari*; and that on at least one occasion the latter name was applied to a cholera epidemic.¹ So far as may be gathered, however, in the great majority of outbreaks of *mahamari* the disease has been plague or a disease practically indistinguishable from it.

The intervals between the successive appearances of plague (*mahamari*) in these districts have been of remarkably varying lengths. Thus, after the first known appearance of the disease at Kedarnath, in 1823, a period of eleven years elapsed before its next recorded outbreak. In 1834-5 the plague was epidemic in the parganas of Nagpur and Badhan Garhwal; and in 1837 it was again prevalent in the latter, along the higher parts of the Pindar river. Then another long period of quiescence follows, and it is not until 1846-7 that it is heard of again. From that time until 1854 there is mention of plague in some part of the districts in each succeeding year. In almost every instance the disease was confined to the hill tracts, but in 1853-4 it descended into the plains and caused an epidemic in the town of Thakurdwara, in the district of Moradabad, in Afzalgarh in the Bijoor district, in Kashipur, and in Rampur. In 1859 it again broke out in the northern parganas, and in 1860 caused a considerable mortality. After 1860 there is once more an interval of ten years in which no mention is made of *mahamari*. But in 1870, 1875, 1876, 1877 and 1878, it was again recorded as prevailing in some portion of Garhwal or Kumaon. In 1884 "typhus fever, identical with *mahamari*" (*sic*) was present in Kumaon, and in each of the four succeeding years *mahamari* was epidemic in some part of one or both districts. In 1889 and 1890, on the other hand, no cases of the disease were

reported. In 1891 six deaths from it were recorded in pargana Badhan. In 1892 and 1893 *mahamari* was also present in the hill tracts, but apparently to no great extent. For the year 1892 the details are lacking, but in 1893 only eleven deaths occurred from the disease. In 1894 there is some doubt whether *mahamari* appeared at all. The Sanitary Commissioner of the North-Western Provinces and Oudh wrote in that year as follows: "The reappearance of *mahamari* in Kumaon in 1892 and 1893, and the report of the occurrence of suspicious cases at Ira, patti Chaprakot in Garhwal, in January [1894], and the report of other cases later, led to an investigation by Surgeon-Major Giles, who, regarding the special outbreak at Garoli, patti Darjuli, in Garhwal, came to the conclusion that the cause of death was not *mahamari*, but gastro-enteritis caused by eating decomposed grain."¹ For 1895 there is again no mention of the disease, though a "very limited outbreak of typhus fever" occurred in Kumaon.² In 1896, the year in which plague first appeared in Bombay, "a few cases of bubonic plague were reported from Garhwal in September, but owing to the flight of the villagers from the infected village, and the unroofing of all infected houses for a period of at least fourteen days, and the disinfecting of all walls, floors, ceilings and house contents with solution of corrosive sublimate, the outbreak was very limited. . . . Experience in the hill district has shown that, although the poison is intensely virulent and sometimes very persistent, it is also intensely local. . . . The above cases were sporadic and quite unconnected with the outbreak of plague in other parts of India."³

Finally, in the spring of 1897 an outbreak occurred at "a small village, near Okhimath, on the main road of pilgrimage from Hardwar to Kedarnath, and some twenty miles distant from the latter place, which was the scene of the 1823 outbreak."⁴ This was apparently the last occasion upon which *mahamari* made its appearance. Plague broke out in that year at Hardwar, at Kankhal and at Jawalapur, but these outbreaks were believed to be due to importations of infection from Sind or Bombay, and not to extensions of *mahamari*.

Such is, in brief outline, the history of *mahamari*, or the hill plague of India. The different outbreaks have differed very greatly in extent and virulence; in some years they have been confined to one or two small, isolated groups of houses, while in other years they have spread over wide areas and affected a very large number of villages. In some years (as in the more recent outbreaks) the deaths from the disease have been numbered by units, while in others they have been reckoned by thousands. The most fatal epidemic seems to have been that of 1853-4, when about 8,000 persons were thought to have died from it. Apparently *mahamari* has become much less formidable within recent years; and since the year 1877, when (at least) 535 deaths from it were recorded,

¹ "Twenty-seventh Annual Report of the Sanitary Commissioner of the North-Western Provinces and Oudh," p. 66. (Also p. 32a.)

² Twenty-eighth Annual Report of the same, p. 10a.

³ Twenty-ninth Annual Report of the same.

⁴ "Government Report on Plague in India, 1896-1897," vol. i., p. 94.

¹ *British Medical Journal*, 1899, Vol. I., p. 1176.

the largest registered mortality in any single year has been 45, and in most recent outbreaks the deaths have been still fewer.

The relation of these centres of plague in the Himalayas to the epidemic which has raged in India since 1896 has been the subject of much discussion. The date of the appearance of plague in Bombay in 1896 has never been determined with accuracy, and even more uncertainty surrounds the question as to whence the infection was derived. In regard to this question only two views are tenable; either the disease developed spontaneously in Bombay, or it was imported from elsewhere. The first view can scarcely be put forward seriously until all evidence pointing to an introduction from elsewhere has been carefully examined and found worthless. If plague was imported to Bombay it must have been imported from pre-existing foci of the disease. In the summer of 1896 plague had been epidemic in Hong-Kong for two years; and other Chinese towns and ports, particularly Canton, Macao and Pakhoi had also been scenes of outbreaks of the disease. The endemic centres of plague in and near the Chinese province of Yunnan were also, as I have already shown, unusually active in those years. The endemic centres in the Himalayas, on the other hand, were showing an almost complete absence of activity at this time. Thus in 1893 there was a small outbreak which caused no more than eleven deaths; in 1894 a small outbreak which was not certainly plague at all; in 1895 no mention of any disease; and in 1896 a limited outbreak—but not until the month of September, when plague had already been present in Bombay for some considerable time, and an outbreak which was definitely stated by competent observers to be “quite unconnected with the outbreak of plague in other parts of India.”

Some facts have, however, been published which seem to point to a possible importation of the plague to Bombay from its endemic home in the Himalayas. The Commissioner of Police for Bombay, Mr. R. H. Vincent, C.I.E., is reported¹ to have stated, in his evidence before the Indian Plague Commission, that in May, June and July, 1896, a large number of *sadhus* (ascetics) came to Bombay from Kumaon. They lived in Mandvi and in a certain temple at Mahalakshmi. They stayed in Bombay for a considerable time, living on the charity of rich *banias*, who ultimately sent them at their own expense to Nassik, a holy place of pilgrimage for Hindus. The mortality among them during their stay in Bombay was said to have been very high. In December a number of cases of plague were found in the Mahalakshmi Temple, where the *sadhus* had stayed while they were in Bombay. It will be observed that there is no proof here of an importation of plague infection from Kumaon; by December it will be remembered, the disease had already established itself throughout the greater part of the city of Bombay, and pilgrims, whether from Kumaon or from elsewhere, would be as likely to catch the plague as other people.

Direct proof of the importation of plague from China to Bombay is, it must be admitted, also wanting.

The indirect evidence, however, of an occurrence of the kind is of considerable strength. It is discussed at some length in the report of the Municipal Commissioner for Bombay for 1896-7 and elsewhere. The principal facts in favour of the view that the Bombay plague came from China are the following: There is constant communication between Hong-Kong and Bombay, and plague infection can, it is well known, be carried by sea over much greater distances than that between these two ports. Then the quarter of Bombay which was believed to have been first infected by plague was that of Mandvi, close to the docks, and “a district full of grain and godowns, and in ordinary times infested by rats.” Further there is the important fact that, though quarantine measures against arrivals from Hong-Kong had been imposed in Bombay in 1894, they were not so imposed in 1896—in spite of the severe recrudescence of plague in the Chinese port in the latter year. It appears that no intimation of the recrudescence had been sent to the Government of India, and it is admitted that it is “quite possible that undetected cases of plague may have arrived from Hong-Kong” and so brought the disease to Bombay.¹

To these considerations must be added the not unimportant one that the plague centres established two years before in Canton and Hong-Kong had already demonstrated beyond doubt their power of giving rise to vigorous epidemics elsewhere. In Macao, in Amoy, in Swatow, and other ports along the Chinese coast, and in the Island of Formosa, outbreaks of plague, which were almost certainly offshoots of the parent stem in either Canton or Hong-Kong, had already occurred. If, on the other hand, the outbreak of plague in Bombay, which has proved to be one of the most virulent kind, with a high local intensity and a great power of diffusion over wide areas, was not due to an importation from China but to an extension of *mahamari* from the Himalayan centres, it is at least remarkable that those centres were themselves in a state of almost complete inactivity in 1893-1896.

The only other alternative suggestions as to the origin of the plague in Bombay are that it came either from Central Asia, or from Mesopotamia or Persia. Each of these possible sources of infection may be considered in turn.

ENDEMIC PLAGUE IN RUSSIAN CENTRAL ASIA (?).

It is very far from certain that plague exists as an endemic disease in Russian Central Asia. The evidence pointing to its existence there is of the following character. In October, 1898, a very fatal epidemic of plague broke out in the remote highland village of Anzob. This village, which was almost the only place affected, lies very close to the Russian border, at the foot of what is known as the Anzob Pass, which divides the Province of Samarkand from the Bekdom of Gissar in the Khanate of Bokhara. It is situated about 39° N. latitude and 69° E. longitude, and is very nearly due north of Kabul, and about 300 miles away from that town. I have described elsewhere² the

¹ “Government Report on Plague in India,” 1896-7, Vol. I., p. 110.

² The *Lancet*, September 9, 1899.

¹ As reported in *The Englishman*, December 5, 1898.

principal features of this outbreak and need not dwell upon it more fully here. The interesting point in connection with it is that, while no definite importation of the infection from outside could be ascertained, there was some reason for believing that plague, or a disease allied to it, might be endemic in these remote hill tracts. Dr. Levin, who investigated the outbreak on behalf of the Russian Government, personally examined every surviving inhabitant of Anzob and an adjoining village, and in many he found buboes in various stages of absorption or scarring. In several other persons, who had not been attacked in the recent epidemic, exactly similar appearances were present. These persons said that they had been ill two, four, or even ten or twenty years previously. They described the illness from which they had suffered on those occasions as lasting from three to five days, and as characterised by headache, fever and pain, and swelling in the groin. The swellings suppurated and either burst or were opened by the "wise woman" of the village. That the swellings were not venereal in origin was proved by the fact that children of five or six years of age had them. Neither syphilis nor scrofula was present in a single case, and small-pox could be excluded. Swellings of this kind were discovered in thirty people in the two villages.

In considering these cases Dr. Levin came to the conclusion that there exists in these *kishlaks*, or mountain villages, an endemic disease which may be described as a "*lymphadenitis femoralis suppurativa*." "Might not the plague," he asks, "have been introduced into this remote spot twenty years ago, and have remained to give rise to occasional epidemics?" Epidemics of this kind might, it is pointed out, easily escape notice. The outbreak at Anzob only became known through an accident. The inhabitants of a neighbouring village ran short of linen and sent to Anzob to borrow some; it was only then that they learnt that plague was raging there and that 200 people had already died of it, and it was through them that the authorities learnt of the epidemic. Supposing, it is appositely asked, these villagers had not run short of linen and had not sent to Anzob to borrow some? a whole village might have died out and the world been none the wiser.

For the present it must remain uncertain whether plague is endemic in these hill tracts. If the disease has been present in that region for the past twenty years, it is at least remarkable that it has not on previous occasions caused a severe and widespread mortality such as it gave rise to in 1898. It is urged that an epidemic outburst of the kind might occur without becoming known to the outside world; but an event of the kind must have left a deep impression upon the memories of the highlanders themselves, and some account of it would almost certainly have been elicited in the course of the enquiries into the Anzob epidemic, yet apparently no word of such an occurrence was forthcoming, at any rate from those villages included in the Russian Government's inquiry. At the same time it has to be pointed out that the inhabitants of villages in these remote regions appear to be extraordinarily unaware of even the most appalling events that may be passing in other villages a few miles away, and it is, therefore, just open to

belief that really severe epidemics may have occurred in other villages outside the scope of the inquiry made at the time of the Anzob outbreak.

This is, however, a large assumption to make, and all that can be asserted definitely is that there is presumptive evidence of the endemic existence, in certain villages in this region, of a febrile disease accompanied with buboes; but that proof that the disease is plague is wanting. There is no evidence whatever to show that this centre of disease had anything to do with the outbreak of plague in Bombay and its later extension to the rest of the world.

ENDEMIC PLAGUE IN MESOPOTAMIA.

Mesopotamia has been the scene of several outbreaks of plague. An epidemic is recorded as occurring in Bagdad in 1596, and others occurred in 1773, 1800-1801 and 1830-1834.¹ In 1834 plague disappeared from the country (just as in the following year it disappeared from Persia), and there is no mention of it again in Mesopotamia until 1867 (three years after its re-appearance in Persia).² In that year plague appeared among the Beni-Taraf Arabs on the Hindieh Marshes of the Lower Euphrates, and caused the deaths of at least 300 persons in five villages. After six and a half years' interval it again appeared (in December 1873) and attacked certain places on the opposite side of the river from the scene of the 1867 outbreak, though later it spread across the river to some of the same places as were affected in the earlier year. This time some 4,000 deaths occurred. In 1875 plague was epidemic on both sides of the Euphrates, in a district somewhat to the south of those just mentioned. At the end of 1875 and beginning of 1876 the disease again became widely prevalent in many places in Mesopotamia. Bagdad suffered very severely (4,570 cases and 2,616 deaths occurring in an estimated population of 60,000), and it was believed that in all some 20,000 persons lost their lives in this epidemic. In 1877 plague was again prevalent in Bagdad (but with less severity than in the preceding year), and also in several other places on the Tigris and Euphrates. The recent history of plague in Mesopotamia is imperfect. Nothing has been heard of the disease there in the past few years, and there is no reason to suppose that there was any renewed activity of the plague in this country at the time of its wide extension in China in 1894 or in India in 1896. There is equally no reason to believe that the outbreak in India was due to an importation of infection from Mesopotamia, where, so far as may be gathered from the absence of information, plague had been quiescent, if not altogether absent, for some twenty years.

ENDEMIC PLAGUE IN PERSIA.

Plague has visited Persia in past times even more frequently than Mesopotamia. In the present century it was epidemic between 1829 and 1835 and then dis-

¹ Tholozan. *Histoire de la Peste Bubonique en Perse et en Mesopotamie*, Paris, 1874.

² Parliamentary Paper, C. 2262. "Papers relating to the Modern History and Recent Progress of Levantine Plague," 1879.

appeared for a considerable interval. In 1863 it re-appeared and caused a small epidemic in the extreme north-west corner of the country, in the district of Maku, in Persian Kurdistan. In 1870 it again broke out, on an elevated table-land to the south-east of Lake Urumiah; it prevailed here in a number of villages in the depth of a severe winter. The next outbreak of plague in Persia was in 1876, in the province of Khuzistan, in the south-west of the country. In December of the same year it was prevalent in two villages, Jaferabad and Dezedje, twenty-five leagues distant from the southern shores of the Caspian Sea. In March, 1877, the disease was at Resht, the capital of Ghilan, and close to the coasts of that sea. In Resht and its neighbourhood 4,000 persons were believed to have died from the plague. In September plague was reported from the Khalkal district of Azerbaijan. In the same month it was said to have been present in Astrabad, but the report was unconfirmed; and among the nomad tribes of Herat a disease broke out which presented some resemblance—though far from a close resemblance—to plague. In January, 1878, the disease was again epidemic in villages in Persian Kurdistan, and remained there at least until April. From that date to the present time there is an absence of information about plague in Persia.

In the meantime there is some reason to believe that the infection of plague which, as just pointed out, had reached the southern (Persian) shores of the Caspian Sea in March, 1877, may possibly have been carried some months previously to the western (Russian) shores of that sea. In Baku three deaths occurred in November, 1876, all in the same house and all after a very short illness, characterised by fever and the appearance of a bubo. They were not followed by any further cases, but after the spread of the disease to the Volga these deaths were recalled and believed by good authorities to have been due to plague.

In the autumn of 1877 a very similar disease appeared among the Russian troops in the Caucasus engaged in operations connected with the Russo-Turkish war. The late Count Loris Melikof was in command of those troops, and a year-and-a-half later he was appointed to take entire charge of the plague measures in the government of Astrakhan after the outbreak of plague at Vetlianka. It was when holding the latter appointment that he recalled that a disease, which was described at the time as "typhus fever with buboes," had, in 1877, caused a high mortality in the troops under his command in the Caucasus. The medical authorities in St. Petersburg¹ came to the conclusion that in all probability this disease had been plague, and apparently plague of a far from mild form, as the case fatality was from 75 to 80 per cent. Further evidence in regard to this disease has been published by Dr. Galanin, whose monograph on "Plague"² is of special value for its account of the disease in Russia and adjoining countries. This author was himself for eight months with the Russian

army under General Gourko, from the third attack on Plevna to the time of the Treaty of San Stefano. He was not in the Caucasus (where the bubonic disease occurred) but in European Turkey, and he points out how easily cases of plague might have occurred without their being recognised by the army surgeons. Cases presenting difficulty of diagnosis, cases regarded as either typhus, or relapsing, or malarial fever, or as some form of croupous pneumonia, were met with, "but," he writes, "it never entered into anyone's head to examine the lymphatic glands or to give its proper significance to a bubo—syphilis, and particularly chancres, being very common in an army in the field—and this was so simply because the significance of these symptoms was, for us surgeons, literally unknown."

It may therefore be taken as probable, though not as absolutely proven, that plague was active in the Caucasus, and as possible, though still more remote from actual proof, that it was present among the Russian troops in European Turkey in the autumn and winter of 1877-78.

In the summer of 1877 a disease of which fever and buboes were the principal features was prevalent in the town of Astrakhan and in a number of villages around. These cases have been regarded as of the nature of a mild form of plague or *pestis ambulans*, and they form an important link connecting the outbreaks in Persia, and particularly that in Resht, with the epidemic on the Volga.

The epidemic on the Volga was (probably) the furthest extension of plague from its endemic centre in Persia. I have elsewhere³ described this outbreak in some detail, and do not propose to dwell upon it at length here. It was confined to six villages on the banks of the Volga, and lasted from October, 1878, to January, 1879. In all, some 515 cases and 431 deaths occurred here. The outbreak was self-limited and came to an end before any organised measures for its extinction were put into force.

A SKETCH OF THE LEPER ASYLUMS, BRITISH GUIANA.

By F. A. NEAL, M.B., C.M.
Medical Superintendent.

THE Government of this Colony need yield the palm to none in its care of the sick and poor. Whether the policy has been altogether a wise one for the social welfare of the people is another story; the fact remains that for its population perhaps no country in the world has so many or such large hospitals, asylums or almshouses as British Guiana. The care of the leper has been no exception.

During Slavery each plantation provided for its own lepers, who were relegated to the back dams of the estate, and though means appear to have been adopted to cut off the diseased from the healthy, leprosy was common even then, for one finds in an archive in the Colonial Secretary's office, Georgetown, October 22,

¹ *Protokols of the Society of Russian Physicians in St. Petersburg*, 1879.

² "*Bubonnaia Tchuma*" (Bubonic Plague). By Dr. M. J. Galanin, St. Petersburg, 1897.

³ *Indian Medical Gazette*, September and October, 1898.

1831, in a "Return of Leprosy Negroes in the Esse-
quibo Division of the United Colony of Demerara and
Essequibo," the large number of 431, a proportion
which if continued would give something like 5,000
lepers in the Colony at the present day, a number far
in excess of the actual.

The first mention of a Government Leper Asylum
occurs as long ago as 1832. On May 1 of that year
the lepers were removed to an establishment on a
sand reef near the mouth of the Pomeroon river and
in proximity to the Indian Post. This "Post" was
under the Post Holder, or Protector of Indians, and
all accounts for the maintenance of the lepers were
included in those for the Post. The Indians do not
appear to have appreciated this attention, neither
could the buildings of the settlement have been of
a substantial character, for a few years later the Post
Holder wrote of the "ruinous condition of the leper
establishment, recommending its removal a mile
further, as the Indians had manifested an inclination
to leave the Post." There appears little need for this
despatch, for we find in a report by the Inspector
General of Police that on his visit there in May, 1839,
he found *five* lepers only. As he arranged for in-
creased accommodation, this number must have been
augmented, though for a brief period only. An
interesting feature attaches to this settlement, how-
ever, as being the alleged cause of the propagation of
leprosy among the warrior Indians—the tribe that
associated more intimately than any other with the
lepers.

In 1845, an ordinance to establish receptacles for
lepers, and provide for their care, maintenance, and
support, was passed. No mention is found as to
where these receptacles were. From some of the
older people one gathers that at an early period—it
may have been in slavery days—a leper settlement was
formed above Hyde Park, up the Demerara River,
and probably on the Himaroonie Creek, which to this
day is known as Cocobay (local name for a leper)
Creek. It is certain, however, that shortly after the
passing of that ordinance, at any rate from 1850,
lepers were located in the Colonial Hospital, George-
town, in a part known as the Lazaretto, and which
consisted of a low one-storey range running along
Thomas Street, and on the site of the pile known as
the Manget Wards.

In time this lazaretto became too small for the
numbers seeking admission, and the almshouse had
to accommodate them. To obviate this a small estab-
lishment was built in what is now the Bourda district
of Georgetown, somewhere about the end of Regent
Street. These people must have been in evidence
in the streets of Georgetown in those days; and it is
not surprising to find the Lazaretto and Bourda
establishments were abolished and their occupants
sent, in 1858, to the newly acquired buildings in the
country at Mahaica.

Kaow Island, in the Mazaruni River, near its
junction with the Essequibo, opposite H.M.'s Penal
Settlement, and about sixty miles from Georgetown,
was probably one of the receptacles of the 1845
ordinance for males, and some say the neighbouring
Calf Island for females. This cannot be vouched for;
but a black man, Orion Mason, who was admitted in

1853 into the Lazaretto, Georgetown, informs me
that he remembers well some of his fellow inmates
speaking of Kaow Island; how a number of them,
owing to bad treatment and neglect, left the island in
two open boats with the intention of coming to town,
were picked up exhausted on the west coast and sent
on to the Lazaretto. Orion was admitted into the
Mahaica Asylum at its opening in 1858, and though
not now an inmate, is still a hale hearty old fellow,
dependent on no one.

In December, 1870, an ordinance to make provision
for the care and maintenance of lepers passed the
Court of Policy, and Kaow Island was again taken
up on the recommendation of the Colonial Surgeon-
General, Dr. Manget, to enable Dr. Beauperthuy to
carry on his experimental treatment. For this object
huts were prepared on the island, and a number of
selected cases, all males, were forwarded from
Mahaica. Beauperthuy himself lived in a wooden
hut at Bartica Point, on the mainland, but unfor-
tunately died soon after settling there.

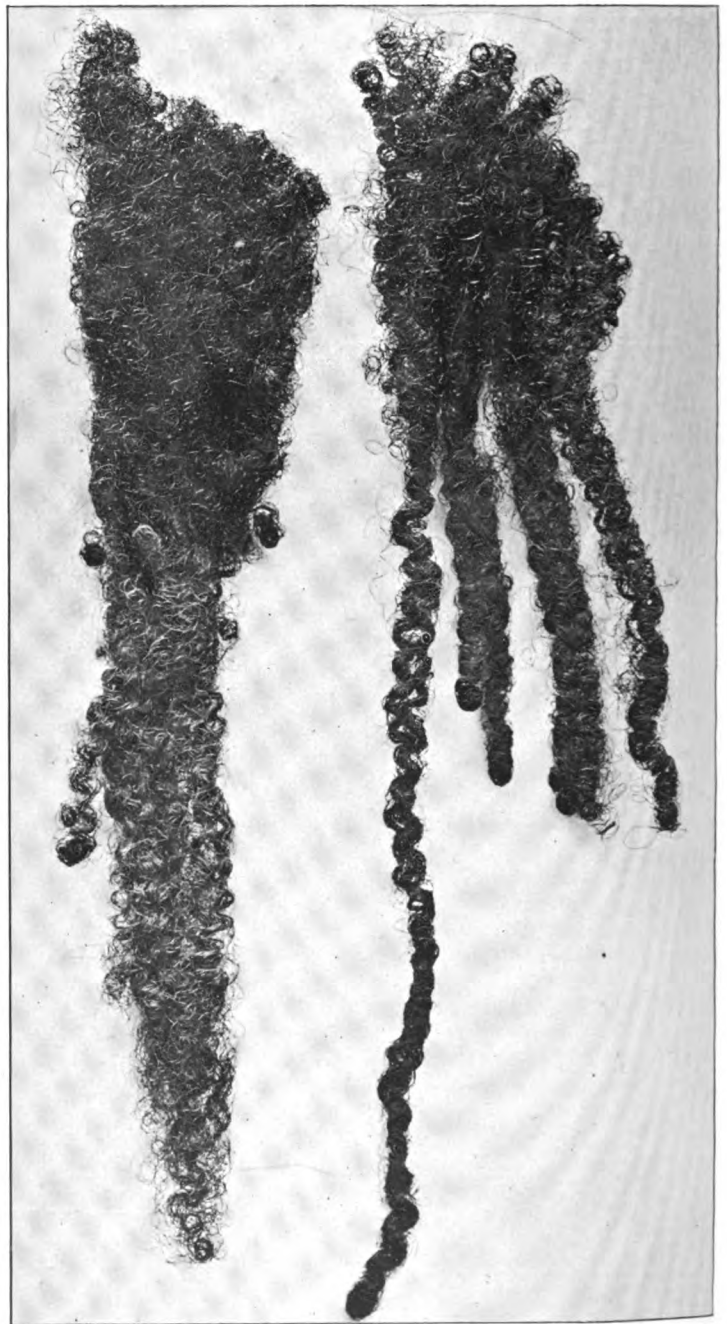
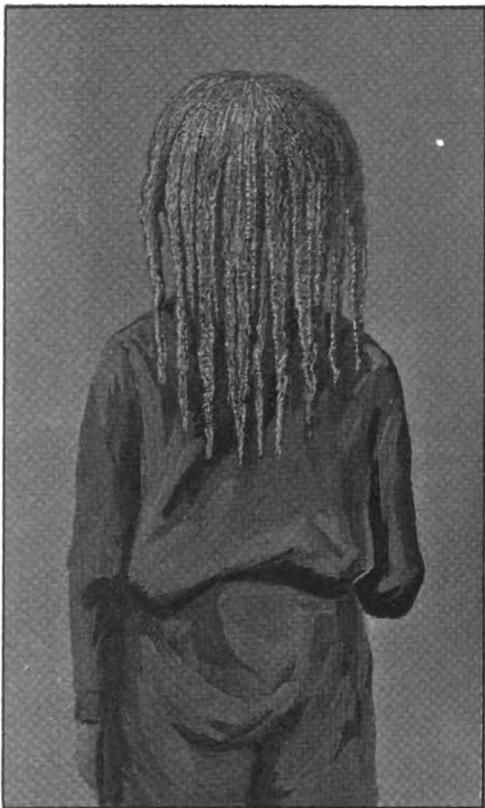
The Kaow Island Asylum was under the Super-
intendent of H.M.'s Penal Settlement, the surgeon
of which was the visiting medical officer. After the
death of Beauperthuy it appears that no further
experimental treatment had been tried, and this
really was the cause of its regeneration. So, why it
was continued as a separate establishment, when the
more convenient one at Mahaica already existed, is
not easily understood. In 1874 the Government,
however, woke up to this fact, and the Special Com-
missioner for visiting the Leper Asylums, Dr. A. G.
M. Cameron, was asked to give an opinion "as to
the advisability or otherwise of retaining the asylum
on this island any longer, either as an experimental
or convalescent leper asylum as at present, and
whether it would be advisable to transfer these lepers
to Mahaica, or to send the male lepers from Mahaica
to Kaow Island, having only one establishment for
the reception of these unfortunate people." Dr.
Cameron recommended removal from Kaow Island,
and so little was done towards the maintenance of the
buildings, which were really huts with wattled walls
daubed with mud and mortar.

In the report for 1879 of the visiting medical officer
to the Superintendent of H.M.'s Penal Settlement,
he states: "The walls become cracked in dry weather,
and when the rainy season sets in, large pieces often
fall down, leaving apertures which freely admit of wind
and rain."

Again, in 1882, the Inspector of Prisons, in his
annual report on Kaow Island, pointed out the ruin-
ous condition of the huts, and in the following year
the medical officer adds the finishing touch: "The
patients' huts are sadly out of repair, and if not im-
mediately taken down, will perform that operation of
their own accord."

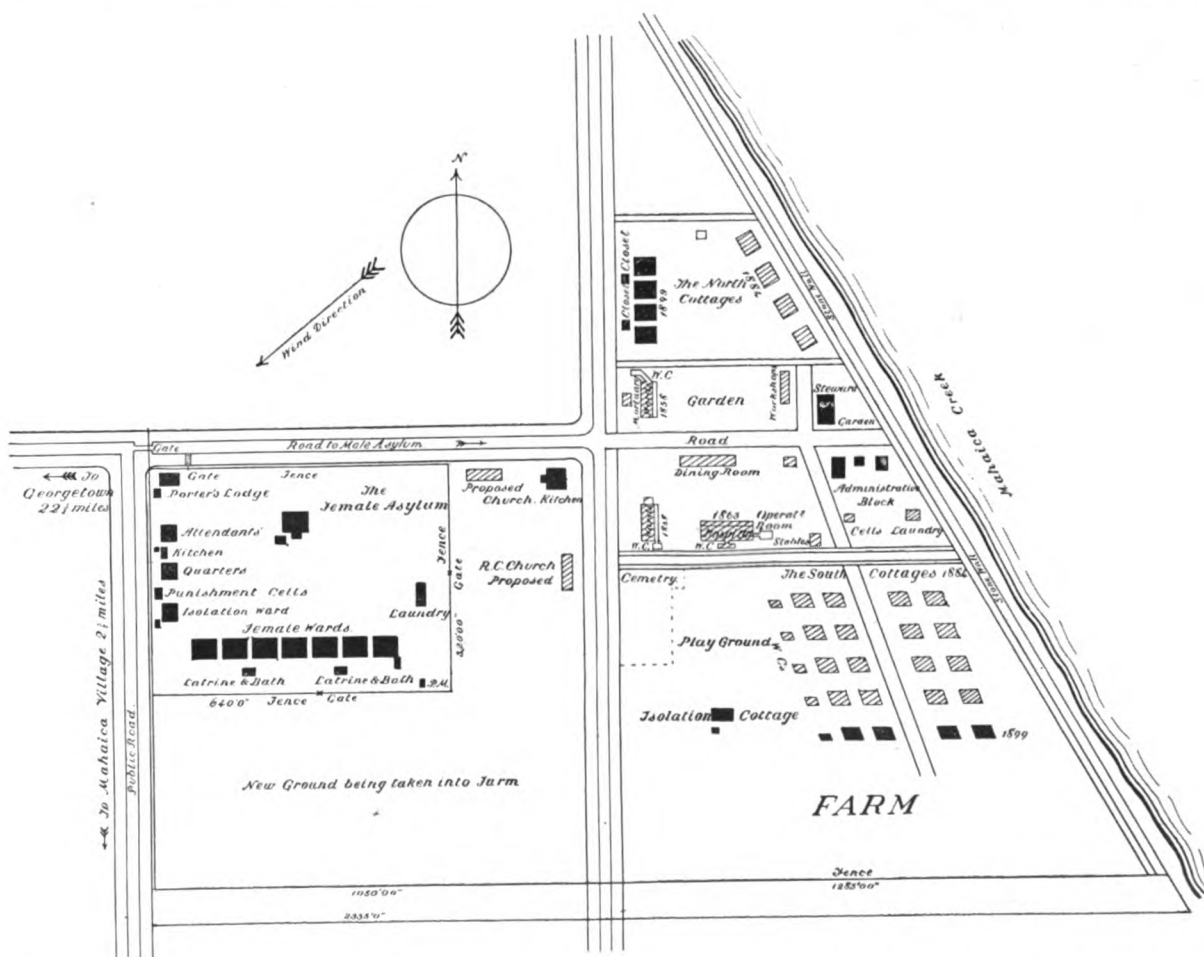
It was not till ten years after Dr. Cameron's re-
commendation that it was carried out, and the inmates
received into the Mahaica asylum on October 27,
1884. They were brought down by a party of police
under an inspector, who, before leaving, set fire to the
buildings so as to prevent people from squatting, "and
so becoming infected with leprosy."

It has already been noted that an asylum existed



ABNORMAL HAIR GROWTH IN A NEGRO CHILD, NATIVE OF TRINIDAD.

Illustrating Dr. R. C. BENNETT's Article published in April number, 1900.



GENERAL VIEW OF FEMALE LEPER ASYLUM, BRITISH GUIANA, AND PLAN OF LEPER HOSPITAL.

In Illustration of Dr. F. A. NEAL's Article published in April number, 1900.

at Mahaica. Under an ordinance passed in 1858, the premises, with buildings recently occupied as a military post on the west or left bank of the Mahaica Creek at its mouth, and about 2½ miles from the village, were purchased from the Imperial Government. It was opened on May 29, 1858, with 77 inmates, 66 males and 11 females.

The buildings then consisted of two one-storey barracks, each 72 ft. × 24 ft., with an open verandah 8 ft. wide in front and raised on 6 ft. brick pillars. The staff consisted of a resident lay-superintendent, who occupied the quarters lately tenanted by the officers of the garrison; a dispenser, the dispensary being in part of the superintendent's quarters; a few servants, or guards as they were called, and a visiting medical officer, the surgeon of the neighbouring district. The institution was placed under the control of the Poor Law Commissioners.

From the outset the male side must have been overcrowded, and in 1863 a new two-storey block, now the hospital, 78 ft. × 26 ft. with galleries facing the sea, was erected. This building runs at right angles to the others. At the same time, to increase the much needed accommodation, an upper floor was added to each of the older buildings. These with the long dining-shed form the main block, and are shown in the photographs as they are at the present day. The provisions which were stored under the superintendent's quarters were occasionally damaged during the high spring tides, and about this time, and no doubt to the delight of that officer, a new and raised store room was built. The asylum remained with the buildings described for nearly twenty years, and though there was a large increase in the number of inmates the accommodation was not increased, so that the strictures of Dr. Gavin Milroy do not come as a surprise. Dr. Milroy was sent out by the then Secretary of State for the Colonies to report on the state of leprosy and yaws in the West Indies. He visited the Mahaica Asylum on July 29, 1871, and regarding it in his report says:—"The locality is extremely unhealthy. The wooden buildings of the asylum were formerly military barracks; the health of white troops must have been inevitably quickly ruined by residence in such a spot. It had happily been condemned for even its present purpose before I left England." "The male wards were extremely crowded and ill-ventilated. There were in each ward four rows of beds or rather sleeping places, for most of the patients lay on the floor with but little space between them. At night the atmosphere must have been foul, even though the doors and windows were open. The female wards were comparatively clean and airy." No wonder, for in 1870 I find from the records the numbers were 231 males, 72 females, total 303; and these were housed in buildings in which at the present day we put less than half that number, with improved ventilation and general sanitary conditions.

Further on in the same report it is stated, "that it would be next to impossible to attempt the useful administration of any remedies in an institution that is wholly unprovided with even the most ordinary appliances or convenience for the care of the sick.

The asylum has hitherto been regarded as only an almshouse or place of safe detention for the unfortunates who find admission into it. On leaving the buildings I could not but feel that the care of the leper of the present day is much like that of the poor lunatic of the last century."

Before leaving the colony Gavin Milroy was commissioned to select a site better adapted for an asylum. His choice fell on Bartica Point, already mentioned as the residence of Beauperthuy, and as there existed at that time a very reasonable feeling that the sexes should be kept apart, it was proposed that when the male lepers were settled at Bartica Point, Kaow Island should be occupied by the females. Though bowing with all respect to these opinions, writing so long after it is perhaps fortunate Milroy's suggestions were not carried out. Since then, by attention to sanitary matters, improved ventilation, greatly increased accommodation, and by studying the comfort and well-being of the lepers themselves, much has been done to render the place a more suitable one.

For a number of years the inmates had a happy-go-lucky kind of existence, and to remedy this the Governor, in 1876, "was pleased, in exercise of the power contained in Ordinance No. 18, of the year 1870, to make rules and regulations for the control and management of the leper asylums." At this time the responsible head of the asylum was a special visiting Commissioner, whose duty was to pay regular visits of inspection, and to furnish to the Government Secretary an annual report, to which was added the report of the visiting medical officer. The institution was also subject to the inspection of the colonial surgeon-general, medical officer to Immigration Department, and the Chairman of Poor Law Commissioners. With the advent of Dr. Hillis as medical officer, a brighter era began to dawn. His name will remain connected with the leper asylums of British Guiana, not only as the author of that well-known work "Leprosy in British Guiana," but also for his able administration of the institutions in which he took so much interest, and in which he worked for over seven years.

Hillis assumed charge in March, 1877, when the asylum consisted only of the barracks which form the main block now. A quotation from his book will show the state of affairs at the time: "From constant change of medical officers, and the difficulties of obtaining the services of a suitable person as superintendent with the necessary administrative capabilities, the asylum, from want of supervision, had gradually been getting into a discreditable condition, the lepers being allowed to do just as they pleased, and get anything they clamoured for." Further on he states that "the place was greatly overcrowded, the records were imperfectly kept, raw rations were issued to the lepers, and every one cooked his or her food in various parts of the houses and yards. Wines, spirits and tobacco were issued in unsuitable quantities, indiscriminately, and in unsuitable cases, and the general sanitary condition of the place was neglected."

With the view of remedying this state of affairs, Hillis was invested with sole authority, thus becoming the first medical superintendent, and shortly after, in 1880, the office of Special Commissioner was abolished.

There is no doubt that in attempting to remedy "existing irregularities," he brought down a hornet's nest round his ears, which ended in a serious riot on the part of the inmates early in July, 1879, for which some were expelled, others banished to Kaow Island, and the females removed *en masse* to the Colonial Hospital, Georgetown.

As might be expected the stay of the females in town was merely a temporary measure, and on January 17, 1882, they were once more removed to what was for so many years known locally as "The Gorchum." Gorchum was an undrained cattle-farm lying about 5 miles from the Mahaica asylum, on the opposite bank of the Mahaica Creek. The buildings consisted of six cottages, each 48 ft. \times 10 ft. \times 9 ft. to plate, with an open verandah, 8 ft. wide, in front; they were built on 7 ft. greenheart pillars, and were placed in a line so as to get the full benefit of the trade winds. Later, quarters for the resident dispenser, matron and attendants were added, and a few years ago, as the numbers had increased, the galleries were boarded in so as to become part of the ward, and at the same time the area below some of the cottages floored and boarded in for dining-room, church services, and other uses. At the best the Gorchum buildings were of flimsy construction and unsuitable for their purpose, the idea being they were merely a temporary measure, instead of which they were in use for nearly eighteen years.

Since 1863 there had been no additions to the male asylum, but in 1884, as the accommodation had become inadequate for the numbers in the institution, and also to provide for lepers waiting in the Colonial Hospital for admission, and for the inmates of Kaow Island which the Government had decided to abandon, the size of the asylum was materially increased. Fifteen acres of land adjoining and to the south were purchased, and in all twenty cottages were erected, four on the north side of the main block and sixteen on the newly-acquired land. These are built eight on either side of a middle walk, and are on 1½ ft. brick pillars. Each cottage is 30 ft. \times 20 ft. \times 7 ft. to plate, and are placed *en echelon*, each thus getting the benefit of the prevailing breezes. There is a verandah in front and behind; the floors, like the rest of the asylum, are of polished pitch pine; ventilation is good, and each could accommodate with comfort eight people. Taken as a whole this cottage system is, to my mind, the ideal of a leper home (see photograph).

By the Medical Ordinance of 1886 the leper asylums came directly under the control of the surgeon-general, and the dual office of district medical officer and medical superintendent abolished, each being placed under separate officers, Dr. Castor being the first whose duties were purely of an administrative character. This change was essential for the proper supervision and working of a large institution and a peculiar people.

Even with these additions and changes Mahaica as a site did not hold a secure position, for somewhere about 1891 Liberty Island, low down in the Essequibo River, was taken up and some money spent in clearing up the bush. However, in 1893, a committee was appointed to report on the working of the asylums, and the advisability or otherwise of retaining the male and female asylums on their present sites. The com-

mittee recommended the retention of the male asylum and the removal of the female lepers from Gorchum to Mahaica. For many reasons this was advisable. The administration of Gorchum from the male asylum, a distance of 5 miles, was practically difficult; its situation, near a village and sugar estate, objectionable, there was no means of keeping the women within bounds, and the buildings would have to be renewed in a few years. A sum of 25,000 dollars was voted for a solid stone sea defence, and another sum of about 100,000 dollars for the purchase of land and erection of suitable accommodation.

Twenty-two acres of land adjoining and to the west were acquired, and on a portion of this the buildings for the female lepers were erected, and they were removed to their new home on September 9 of this year. There are seven cottages as against six at Gorchum, each with a gallery eight feet wide both back and front, and built on nine feet brick pillars. The area below each is concreted and at present the central cottage only latticed in below as a dining hall. Others can be similarly dealt with as required. There are the usual bath rooms and lavatories—for the hospital these are on the ward level, but detached in the rest. Other buildings on the female side are a laundry, handsome quarters for matrons, and the female attendants will no doubt appreciate their new quarters after Gorchum. The photograph taken before the fence was put up shows most of the buildings in the female department, with the additional eight cottages on the male side, similar in plan to the existing ones, but raised on six feet pillars, the accommodation is now ample for about 500, without overcrowding. Other additions consist of improved store, attendants' mess room and hospital, new dispensary, office, kitchen, operating room, and resident officers' quarters, all now building; and the whole asylum of forty-five acres is enclosed in except on its sea and river face by a fence of thick zinc sheets eight feet high, and the female portion an enclosure of seven and a half acres within the general enclosure.

Such then is the history, no doubt incomplete, of the leper asylums of British Guiana, and more especially of Mahaica, which from a small beginning forty-one years ago, ranks most probably as the largest voluntary asylum in the world. To use the words of some of the older inmates who knew the place in its infancy, "It was a nigger yard in those days but now it is a town." The numbers are about 400—to be precise 407—admission is voluntary, except when lepers expose themselves, or in any way become a nuisance; and all are expected to conform to the rules and regulations. The inmates consist of black and coloured Creoles of the Colony, a few Africans, Portuguese, sprinkling of Chinese, and a large proportion of East Indian Coolies.

With its churches, and its schools for the younger ones, with its different workshops for turning out shoes, clothing, carpentry, &c., its work in the farm and yards, and the dozen and one in- and out-door occupations always to be found in a large institution, the asylum is like a village community in which it is the desire of those in charge to consider the comfort and well-being of these unfortunates, and to give them as much of home life as possible.

The photograph and plan are amateur work, but will, I trust, help to give a clearer idea of the leper asylums of British Guiana than a mere bald written description alone.

PECULIAR CONDITION OF THE HAIR.

By R. C. BENNETT, M.B., C.M.

Government Medical Officer, Trinidad.

THE accompanying photographs showing specimens of a peculiar condition of the hair in a negro child are sent with this contribution on a subject which, though of no clinical importance, may be interesting.

The condition of the hair when viewed *en masse* is striking and peculiar. It grows in rather a luxuriant manner, and separates itself with rope-like and attenuated strands quite separate from one another, and made up of closely interwoven meshes. The strands measure from six to twelve or more inches. The parts of the strands nearer the skull are black in colour; the distal ends are a pronounced red, due to exposure to the sun. These flax-like strands, bunched together, give a peculiar and characteristic appearance to the individual. This condition of the hair seems to be a "congenital" one. I have seen many cases, always in very young children—never in adults.

It has been suggested that the condition of the hair is due to dirt and neglect. This is not so, and there are no grounds for believing that it is, and the specimens forwarded do not indicate that. The boy from whom these specimens were obtained had a twin brother whose hair was in exactly the same condition.

What the nature or pathology of the affection is, I have had no time recently to inquire into. Much superstition prevails amongst the natives affected, and the parents positively refuse to allow the application of the scissors to the affected hair, so that the child has to carry this abnormal mass until he is old enough to cut it away *with his own hand*. It was with difficulty that I obtained either a specimen or a photograph in this case. I think the affection has been described and named by some French writer—"Plic"? I have never seen it in other but black children. I do not think it is due to disease of the hair follicles, but to some abnormal condition of the individual hairs causing a twisting and inter-twining. I have grounds for believing that after the hair has been cut away, the condition does not recur.

The snapshot photographs I enclose I fear are rather bad, but they were the best I could obtain. The specimens of hair are, however, characteristic, though no indication of the length to which they grow.

TRACHOMA—AN OINTMENT FOR.—R. Copper sulphate, salicylic acid, cocaine, of each 1 part; vaseline, 100 parts.—*Deutsche Medizinische Zeitung*.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By GEORGE H. F. NUTTALL, M.D., Ph.D.

Pathological Laboratory, Cambridge.

(Continued from p. 200.)

OPINIONS, however, do not agree as to the influence of trees upon malaria. Celli (1900, February 10), in fact, holds quite the opposite opinion, as do also other Italian observers. Celli claims that "Aboriculture of a malarial territory is favourable to the development of malaria." Investigations in Italy have moreover certainly shown that one species of malaria-bearing mosquito (*Anopheles bifurcatus*) occurs in woods. Celli considers that the *Eucalyptus*, *Coniferae* and *Ricinus communis* have in this respect been "the subjects of groundless praise."

Bodies of water lying in the course of winds coming from a malarial centre are known to be protective. Ships anchored off a malarial shore where the wind is blowing from the land, remain free from malaria unless they get quite close in, and even then the cases of infection are rare.

Sir John Pringle ("Observations on the Diseases of the Army in Camp and Garrison," London, 1752, cited by Laveran) states that the British army in Holland in 1747 suffered so severely from malaria that some battalions could only muster a seventh of their men. The squadron anchored in a canal between Zuit-Beveland and the Island of Walcheren, was quite free from fever.

Blane ("Observations on the Diseases incident to Seamen," London, 1799, p. 221, cited by Hirsch, p. 209), wrote: "When the ships anchored at Rock Fort, they found that if they anchored close to the shore, so as to smell the land air, the health of the men was affected, but upon removing two cables' length,¹ no inconvenience was perceived." Rattray (*Edinburgh Medical Journal*, 1859, February, pp. 708, 710, Hirsch) made similar observations on ships lying in Hong-Kong harbour: "The fever . . . while fatally prevalent on shore, the ships in the harbour, even when lying at very short distances from the shore, are usually or often exempt from its ravages." Vincent and Burot (*Le Paludisme à Madagascar*, *Rev. Sc.* 18 juillet, 1896, et *Acad. de Méd.* 7 avril, 1896) write that the most of the soldiers in the Madagascar expedition acquired malaria whilst the sailors on men-of-war or merchant ships, in spite of great fatigue, remained unaffected. Some vessels remained as long as six months at the anchorage of Majunga, scarcely 300 metres distant from the shore, but none of the men, with the exception of those who were sent up the river and were obliged to sleep on the ground, acquired fever.

This indicates that the infectious agent is heavy and must gravitate into the water. Now we can readily imagine that mosquitoes, which are not capable of maintaining a prolonged flight, fall on to the water, and, especially if it is agitated, may not be able to rise again. Besides, if they rise it would rarely be

¹ A cable measures 120 fathoms. Two cables would represent an increase of 480 yards in the distance of the ship from shore.

to the height of a ship's deck. Assuming that the mosquito is the carrier of the infection, we can understand how the wind passing overland would carry the infection further than on water, for the mosquito would rise (this occurs especially during the lulls in the wind) and fall continuously in one direction, being able to rest occasionally, his powers of further flight being unimpeded. I have often, and others have also, no doubt, seen insects rise and fall this way in the air, always being carried some distance in the direction of the air current. The flight of the mosquito being usually close to the ground, and its frequently having to stop and rest by clinging to the vegetation, would account for the insect being usually transported but to a limited distance, and explains how the woods serve as sieves to the "malarial poison." (When the wind is blowing strongly mosquitoes are no longer troublesome, because they seek shelter and cling to the vegetation.)

Cultivation of the soil has hundreds of times been observed to rid a district of malaria. Hirsch cites a number of cases (p. 193), see also Laveran (1898). As King and others have observed, this is probably due to the swamps and pools, the "mosquito-nurseries" being drained and removed. I should add that a change in the character of the vegetation might also be considered a factor in relation to these insects.

Flooding the Land.—The complete flooding of pools, ditches and marshy land has proved as effective as draining the land and cultivating it. (Hirsch, p. 193), Dods (1878), who spent twenty years in the tropics, had never seen malaria particularly prevalent in the rice fields as long as they were covered with water; it is only when the crops are cut and the rice fields begin to dry up that they are dangerous. Laveran (1898, p. 31) cites Boileau-Castelnau (1850) as stating that rice fields are healthy when the water circulates, but the reverse when it stagnates.

The avoidance of Sleeping out of doors at Night, or of Exposure after Sunset.—It is notorious that malaria is most dangerous when the sun is down, whereas it seems relatively inert during the daytime (Laveran, 1898, &c.). King writes, "With regard to the mosquito, however, it is well known that it remains, for the most part, during the day harboured in woods, weeds, or low underbrush, and comes out after sunset and at night to indulge its blood-sucking proclivities." It is well known that sleeping out of doors after sunset is more dangerous than waking, "for it is undoubtedly true," writes King, "that while awake, the person exposed will move about, or brush away the insect, while he will submit to be bitten during sleep." Bignami (1896) makes the same statement. In this connection a communication by Home (October 28, 1899, p. 1191) is of interest as an example. In January, 1887, the *Turquoise* was at Kilwa Kisiwani, East Africa. Three officers and seven sailors camped on shore one night, after having been in search of a lost sailor. The men made a large fire, but were nevertheless greatly troubled by mosquitoes. The officers remained awake all night finding it impossible to sleep, whilst the sailors did not allow their slumbers to be disturbed. The ship afterwards sailed to Zanzibar. On the way, that is after about ten days, all the sailors developed malaria, whilst the officers remained healthy.

Home says that he has found six similar cases in the health-reports of the British Navy. Bromlow refers to this case in the "Blue-book" for 1887 (pp. 80-81) as follows: "The only difference was—the officers did not sleep, the men did; there was no great fatigue undergone except in the case of the lost sailor man; the men lay on, and were covered with, canvas."

The Use of Fires.—King writes, "In malarial districts the use of fire, both in-doors and to those who sleep out, affords a comparative security against malarial disease." . . . The mosquito "is well known to be attracted by lamps, lights and fires into which it heedlessly flies at the cost of its life." . . . "Every fire, therefore, whether in-doors or out, is a sort of mosquito Hades. In some tropical countries, despite of heat of climate, fires are kept up all night in every apartment as a preventive against fevers; and experience has demonstrated that they are more effective when placed between the open window (or door) and the body of the person to be protected. In this way it is easy to comprehend how every mosquito will fly directly into the light and the fire before reaching the thus protected sleeper." The smoke itself would also drive away insects. Bignami (1896) says that those persons in the Roman Campagna who sleep in shepherds' tents (which are cone-shaped, with a fire in the middle of the floor and an escape for the smoke at the top—the atmosphere in the interior being very smoky) do not acquire malaria. I have already referred to the fires used by the fishermen in the Punjab. I should say that the ascensional currents of air produced by fires will also carry many mosquitoes away.

Immunity of Persons working in Sulphur mines, Fumigation said to afford protection.—d'Abbadie (1882) states that the native elephant hunters in Ethiopia who reside on high plateaus where the climate is relatively cool venture unmolested into the hottest and most malarious districts. "They attribute this immunity to their habit of subjecting themselves every day to fumigations of sulphur," the naked person being exposed to the fumes.

d'Abbadie mentions investigations made by Prof. Silvestri of Catania, in Sicily, which showed that the workmen in sulphur deposits ("soufrières") situated at low levels in malarial foyers, were relatively exempt from fever. Whilst 8 to 9 per cent. of the sulphur-workers were malarious, as many as 90 per cent. of the population in the vicinity who followed other occupations suffered from malaria. Fouqué had also informed him regarding the now uninhabited Zephyria near Milo, in Greece. This city had at one time a population of 40,000 inhabitants, but malaria gradually exterminated the inhabitants. It is impossible to pass the night there now without acquiring malaria. The depopulation of Zephyria seems to have begun about the time when the sulphur deposits thereabouts ceased to be worked. Fouqué also wrote that there are sulphur works on the western border of the malarious marshy plain of Catania. Situated near the sulphur works, but on a higher level, are the remains of a village which had to be abandoned at the beginning of this century on account of malaria. A colony of workmen reside at the sulphur works whilst the village above remains uninhabited.

I do not know if similar observations have been made elsewhere. If it is true that fumigation with sulphur protects against malaria we would have another fact to support the mosquito hypothesis, for the smell of sulphur would repel these insects. It seems to me that the matter decidedly deserves further attention. On the other hand mosquitoes would scarcely find a suitable place in which to multiply in localities where the soil is permeated by sulphurous emanations.¹

Racial Immunity.—The relative immunity exhibited by the negro race towards malaria² is due, King thinks, to protective colouring. Besides, many negroes anoint their bodies with grease, whilst others emit an offensive odour from their persons—one or more of these factors may serve to some extent to keep off mosquitoes.³ Laveran (1896) states that delicate-skinned people and children are more susceptible to malarial infection because they are more readily bitten by mosquitoes. Laveran (1898, p. 124) attributes the immunity of the negroes to their thicker skin, and states they are less subject to mosquito bites.

4. *Influence of Occupation.*—"The occupation has much to do with susceptibility to the disease," write Welch and Thayer (1897) "soldiers and tramps who

sleep upon the ground in malarious districts are particularly susceptible. Fishermen in the bays and inlets along the southern shores of the United States, as well as farmers and berry-pickers in the same regions, are particularly open to infection." "Furthermore," writes King, "in certain districts where the so-called 'malarial poison' is supposed to be lodged in trees and bushy plants near the ground, it has been observed that those persons are particularly prone to fever who cut down and disturb these malaria-laden plants, which is extremely suggestive of the mosquitoes being disturbed from their reposing haunts, just as one might get stung by stirring up a bee-tree or a hornets' nest. La Roche, in his well-known work (p. 282) says: 'Malaria is collected by plants, particularly on cutting them down or rooting them up, thus exciting fever in the labourers who might otherwise have escaped, as proved by the circumstance that in all these situations, while the workmen are in the erect posture and engaged at their work, they escape the fever, but are attacked if they sit, and more particularly if they lie down on the ground—and that whether they sleep or not.' Macculloch (1827, p. 124) says of the Roman Campagna 'if the labourers cut down certain plants (a bushy thistle chiefly), a fever that would otherwise not have occurred, is the consequence.'"

Effect of turning up the Soil.—Malaria may develop in previously healthy districts in consequence of digging the beds of canals, railway tracks, foundations for houses, &c. A severe outbreak of malaria was associated with the excavation of the Panama Canal, and malaria also followed the excavation of the Canal St. Martin and the fortifications of Paris. The same has been experienced many times over in various countries (Hirsch, Welch and Thayer, &c.). It is probable in such cases that pools of water are formed in the excavated land, thus giving rise to mosquito nurseries, and it is possible that these become infected by workmen coming from malarious districts. Moore (*Indian Medical Record*, December 16, 1897) believes that it is a mistake to speak of malaria being caused by "soil disturbance," he says he has always found that there has been an interference with subsoil drainage resulting in "a marsh or allied condition." Poore (1899, p. 461) writes "Surgeon Bowen, R.N., D.S.O., informs me that the turning up of fresh soil is often followed by an influx of mosquitoes."

(To be continued.)

¹ Whilst in Rome in March, 1899, Professor Grassi showed me a letter from an Italian gentleman, which stated that fumigation of the body with sulphur is used in Italy to-day as a safeguard against mosquitoes. Edge (September 30, 1899, *Brit. Med. Journal*, p. 892), I might add parenthetically, found that fleas no longer bit him after he had taken sulphur lozenges. He suggests that persons should thus protect themselves by "saturating their excretions with sulphides, which the fastidious insects abominate."

² See authorities quoted by Hirsch (p. 172). Thayer and Hewetson's (*Malarial Fevers of Baltimore*, Johns Hopkins Hosp. Rep., 1895, vol. v.) figures indicate that the negro is only about one-third as susceptible as the white man. See also cases cited by Laveran (1898, p. 113).

³ That insects are particularly sensitive to certain odours is a matter of wide experience. Whilst the odours of flowers attract some, they repel others, and the same thing may be observed with regard to animals and blood-sucking insects. Fleas (*Pulex irritans*) are repelled by the smell of the horse, and stablemen are protected by the odour they acquire. Railliet in fact advises the use of a horse blanket for the purpose of driving off fleas. The bed-bug is attracted by the smell of the human subject, especially by certain individuals, whilst others perhaps lying in the same bed are not bitten. It is a matter of common experience that some persons are more subject to the attacks of mosquitoes than others. It was noted in the experiments conducted by Grassi, Bignami and Bastianelli (May 7, 1899), that the anopheles preferred to bite two out of five persons experimented upon, and that the persons who were bitten by preference developed greater swellings at the places bitten. Of the three others, two were not troubled through effects following upon the bite, whilst the third was but slightly troubled. Grassi himself was only rarely bitten and then only by anopheles that were very hungry, and in close proximity to which he advanced his hand. This is but another confirmation of what I have said elsewhere that blood-sucking insects behave differently with regard to various persons. King (1883) advises the use for prophylactic purposes (against malaria) of some terebinthinate, camphorated or eucalyptolized ointment or liniment, the use of smoke, &c. Koch, i. and iii. (1898) does the same. Either garlic or camphor, placed in a bag and carried on the person, was considered, as Lind (1779) states, a prophylactic against malaria in the last century, and Laveran (1898, p. 124) relates that the tradition exists in Italy and France that fevers are avoided by eating garlic. Celli and Casagrandi (May 12, 1899, p. 22) report that the peasants in Sardinia, Orosei and Dorgal protect themselves against mosquitoes by rubbing the exposed skin, as also their bedsteads, with garlic.

TREATMENT OF SNAKE BITE.—To be efficacious the treatment must be prompt. If the bite is on a limb put a tight band around the limb above the bite; suck the wound or destroy the poisoned surfaces by cauterisation or by the injection about the wound of permanganate of potassium, calcium hypochlorite, or chloride of gold. If these are not at hand use a caustic alkali, bromine or chlorine water, carbolic, tartaric or chromic acid, tincture of iodine, or even a weak solution of corrosive sublimate. The antitoxine treatment of Calmette should be used whenever and wherever it is available.—CRAW-CRAW.—*New York Medical Journal*, February 17, 1900.

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THE

Journal of Tropical Medicine

APRIL, 1900.

SMALL-POX IN THE PHILIPPINES.

MAJOR C. F. MASON, M.D., U.S.A., in a letter to the *Medical Record* from Iloilo, Philippine Islands, dated December 25, 1899, states that: "Small-pox may be truly said to be universal here among all classes of natives. I doubt if many natives reach the adult age without having an attack."

It is interesting in this connection to recall the position of the Philippines as regards inoculation and vaccination. In an article in the *British Medical Journal*, October 5, 1889,¹ the history of the introduction of vaccine matter into the Far East, was fully gone into, and the question of the potency of vaccination as practised in the

countries adjacent to the China Sea discussed. The Chinese and their neighbours have for centuries practised inoculation against small-pox. Their methods of introducing variolous matter to the human body are as follows: (1) A dried small-pox scab is pulverised and the powder blown up the nostrils. (2) The clothes of a person dead of small-pox are worn day and night for two or three weeks, or until the rash appears. (3) An abrasion on the arm or leg is smeared with matter from a small-pox pustule. The Chinese, however, do not employ inoculation in any form except during a very bad epidemic of small-pox. Vaccination was introduced into the Philippines by the Spaniards, about the beginning of the Nineteenth Century; and it is interesting to note how the vaccine matter reached a country so distant from Europe. It was brought, not by way of the Indian Ocean, but from South America. Spanish rule obtained in South America and the Philippines at the time, and the Far East was then more in touch with European civilisation by way of the Pacific. It was a long way to bring so delicate a fluid, and it showed the implicit confidence the Spanish authorities had in the treatment. But vaccine was also carried into China and the Far East by the old East India Trading Company, during the first years of the Nineteenth Century. It was brought from India, and at the Merchants' Hall in Canton, a Chinese doctor, A-He-Qua, was engaged to vaccinate his countrymen.

From that day until the present time vaccination has been practised to some extent among the Cantonese. It is only, however, occasionally applied during an epidemic in the case of children, re-vaccination being well-nigh unknown amongst the Chinese, however severe the epidemic. Any one, however, who has observed the pustules raised by the vaccine, generally used by the Chinese, must have been struck with its incapability to cause a "good arm." The Chinese vaccinator usually makes six separate punctures into each arm, and there results a slight local irritation and scabs about the size of split peas. The fact is that the lymph has become so attenuated that it is altogether worthless. It scarcely "takes" at all, and the protection must be

¹ "Inoculation and Vaccination for Small-pox amongst Chinese." By James Cantlie, M.B., F.R.C.S.

infinitesimal. The Chinese have continued to vaccinate from arm to arm for well-nigh one hundred years, either with the variolous lymph originally obtained from inoculated persons, or the vaccine matter introduced by the Spaniards or the British.

It is satisfactory to know that the Far East is better supplied with vaccine lymph than it was ten years ago. Then, all lymph came from Britain or America. The lymph from Britain, owing to the voyage through the tropics, was often useless; but now there is a vaccine institute at Hong-Kong; and from Japan, Saigon, Calcutta, and even Sumatra and Java, lymph is sent to China and the Philippines. Seeing the unprotected state of the natives in the Philippines no doubt the Americans will soon establish vaccine institutes in Manila and elsewhere in the Philippines, and endeavour to induce the ignorant and prejudiced natives to protect themselves against small-pox, without doubt the greatest scourge known in the Far East.

THE TRIALS OF INDIA.

INDIA is passing through one of those prolonged periods remarkable for a series of overwhelming physical catastrophes which history but too frequently records as happening at longer or shorter intervals of time. Earthquakes, floods, drought, famine and pestilence have wrought their havoc on the land, and there is unfortunately no sign of their effects passing away. Famine and plague are extending their boundaries. Already it is reported that five to six millions of people are being supported by the Government and prevented from being starved while plague is passing into those regions where it has been absent so long that men declared it could not possibly come. The greatest sympathy and assistance are due to the Indian people in their unique sufferings and trials, and notwithstanding the exceptional calls on the British at the present juncture we are sure that they will be given. Under the shadow of a great distress there are some bright glimmerings of light which sympathy and

monetary assistance can materially strengthen. The un murmuring stoicism of the people under such adverse circumstances has been simply marvellous, while the herculean efforts of those in authority, in their endeavour to mitigate the suffering, have exceeded the bounds of the most sanguine expectations.

To attain success against famine, efforts have to be directed to one object, which is the distribution of food. If food can be obtained from other localities and conveyed to the places affected and distributed to the people who are suffering, the results of scarcity are counteracted and the inhabitants are kept alive. The problem is clear and uncomplicated, and provided the extent of country affected is not too great, and the prolongation of the famine does not continue for a number of consecutive years, it can be resolved by money, organisation and means of transport. The Indian Government have fought against the famine in a most extraordinarily successful manner, achieving results far beyond those of any other Government face to face with so great a calamity. We trust that their fight will not be in vain, and that neither the extent nor the duration of the famine will become of such magnitude as to render it impossible for human endeavour to cope with it.

If success, though difficult to attain, is within the limits of possibility because the problem to be resolved is understood, famine in this respect is an easier task for the Indian Government than the plague. Concerning the latter we know much more than ten years ago, thanks to the work of Kitasato, Yersin and Haffkine, but unfortunately, when our knowledge is all summed up it does not amount to very much. Plague spreads, notwithstanding the preventive measures which India has adopted. If the measures recommended are sufficient their failure must rest on their administration, which is not improbable, for the medical organisation to meet such a pestilence is utterly inadequate in India. That is one aspect of the question, but another, and perhaps for the time being the most important, is the contentedness with which the authorities continue to tread in the old ways, and leave

the elucidation of the problem relating to the spread of plague to chance, instead of making it their primary object. During the five years in which plague has prevailed in India there has only been one attempt to make anything like a scientific investigation into the subject, and that was in the first months of the plague in Bombay, when a committee of research was appointed. This apathy is not creditable to a Government which is losing thousands a week of its inhabitants from plague for lack of knowledge concerning the disease. We can only imagine it is due to a want of grasp of the fundamental fact that the prevention of disease must necessarily rest on an exact comprehension of the methods by which the disease spreads, for otherwise, the action of a Government which is winning golden opinions for its famine work is inexplicable.

Article for Discussion.

DOES CANCER OCCUR IN NATIVE RACES RESIDENT IN THE TROPICS? IS SARCOMA KNOWN?

THE statement has been so often made that cancer is unknown amongst races of people leading a "natural" life that it is time the question was settled one way or the other. The letter published elsewhere in this issue from Dr. John Haddon is pertinent to the question. Dr. Haddon ascribes the increase of cancer amongst British folk to the fact that they are daily becoming more and more a "meat" eating people. Whether it be the case that cancer is actually increasing or no, or whether it be the case that the eating of meat contributes to the tendency to malignant disease, there seems little doubt that cancer is very prevalent in Britain. In taking up this discussion, a most important one practically and scientifically, I would urge the importance of notifying two or three points so as to obtain exact knowledge. It is generally believed that sarcoma does occur amongst native tropical races, but that cancer is rare or even unknown. Therefore, in any communication on this subject, it is to be hoped the

distinction between these varieties of malignant disease will as far as possible be made. Where, however, means of microscopic diagnosis is impossible, it will add to our knowledge greatly to have the fact of "malignancy" notified alone. Again, it is important to be exact as to the nationality of the case recorded. I have frequently had occasion to operate on Eurasians (half castes) for sarcoma and less frequently for cancer, but the cases of malignant diseases in pure natives known to me have been much fewer, and have been of a sarcomatous nature as far as my experience goes. A third point of importance to note, in recording malignant cases, is the nature of the diet. Many natives, more especially those who come for treatment to a European doctor, have partly or wholly adopted European food as their diet. It is evidently important, in view of the assumed diet origin of cancer, that this point should be noted. Where statistics as to the relative proportion of cases of malignant disease to the population are available they would serve a useful purpose.

In answering these questions, therefore, there are at least five points to be dealt with. (1) Does cancer occur in your practice amongst natives? (2) Does sarcoma occur? (3) Do they occur amongst Eurasians (half castes)? (4) What is the nature of the diet of persons afflicted? (5) What proportion of the population is attacked by malignant disease?

J. C.

Replies to Articles for Discussion.

PREVALENCE OF SCARLET FEVER, &c.

SCARLET FEVER is said to be unknown in this Island; not a case has been reported to the Board of Health.

Smallpox was "stamped out" a year ago by 792,000 vaccinations, performed in three working months, with virus produced on the ground; this is the greatest record ever made in vaccination in *three months*.

The few lepers found here have been gathered

into a hospital; the disease has been known now for fifty years, but shows no tendency to spread.

After the great hurricane of August 8, 1899, there was a mild epidemic of dysentery, easily managed.

Anæmia is the scourge of the Island at the present time; it is believed to be due to the *Ankylostomum duodenale*.

GEO. G. GROFF.

San Juan, Porto Rico.

REPORT ON THE HEALTH OF CHEFOO FOR THE HALF-YEAR ENDED SEPTEMBER 30, 1899.

By JOHN FRANCIS MOLYNEUX, M.R.C.S., L.R.C.P.ED.

Cases of Intestinal Hemorrhage.

IN my last Report,¹ attention was drawn to the prevalence during the latter part of September, 1898, of intestinal hæmorrhage; again this year we have experienced a somewhat similar but more grave visitation. Upon its first outbreak I interviewed the late Dr. Douthwaite (whose untimely death, after long, skilful, and generous service to both foreigners and Chinese alike, this port has to deeply deplore). Dr. Douthwaite stated that in the China Inland Mission Schools he had, in the past, occasionally met severe but rarely alarming cases. He described the condition as "colitis," pointing out the absence of some marked dysenteric symptoms and especially noticing the characteristic fetid odour of the stools. He further pointed out that in his experience relapse was very common, and that an attack often lasted over a period of many weeks. Less than a month after our first interview my lamented colleague died of the same malady which he had so carefully investigated.

Three very severe cases have been under my care. Two are now convalescent (adult female, aged 19; male child, aged 7), but in each instance there was very grave danger, and convalescence was long deferred.

The third case terminated fatally after about three weeks' illness. The patient was an adult male, aged 52, of very powerful build and fine physique generally. An irregular diarrhoea, one day more, one day less, ushered in the attack. The first stool seen by me contained blood in quantity, and the patient was at once sent to bed. There was no tenesmus, no temperature, no pain. Thirty-five grains of ipecacuanha powder was exhibited, after which he passed the only satisfactory motion seen during his illness. Two days later I repeated the ipecacuanha in the form of the fluid extract, giving a full dose.

The bowel was washed with tannic acid in warm water (3 grains to an ounce), ordinarily twice a day. Later, hazeline was injected into the bowel in place of tannic acid. Both, beyond lessening the frequency of evacuations, had apparently little effect upon the

hæmorrhage, which became so alarming that I had to use hæmostatics in large doses—diluted sulphuric acid, tincture of opium, and extract of liquid ergot in combination (the opium against my will, but it was necessary)—and later, after the case had been seen by the senior fleet surgeon on the station (Dr. Sibbald), hypodermic injections of ergotin were administered. Cold compresses (the port had run out of ice supply) had no effect. Strychnia and other cardiac stimulants were exhibited as the pulse became somewhat intermittent. On October 15 I found the patient at mid-day somewhat delirious, and was informed that he had been so for some hours. He was seen at 10 p.m., and the pulse was fairly good; between midnight and 1 a.m. I was hurriedly sent for and found the patient *in articulo mortis*—pulseless, and covered with a profuse cold sweat. Strychnine and brandy were hypodermically given, but without effect, and the patient died in about two hours.

The sudden collapse was due to a fresh and most extensive hæmorrhage. After death, during the preparations for encoffining the deceased, large blood clots were found beneath him, and upon being moved, blood in extraordinary quantity was extruded. That extensive hæmorrhage was going on had been obvious for some days; but in my experience at Ningpo, in 1894, when the Chinese camp suffered from dysentery at Changhai, and hundreds passed through my hands, I have seen no hæmorrhage to compare with this case. Throughout the attack there was never any symptom or indication of perforation or peritonitis.

A fatal case of "colitis" occurred in the China Inland Mission Boys' School. Dr. Douthwaite found a tear in the colon, which had been caused during convalescence by the child indiscreetly getting up quickly and walking across the room.

In my two severe cases (now convalescent) I found that so soon as the motions became at all hardened there was a consequent reappearance of blood.

The diet is naturally an all-important care; all milk, etc., was zymised and so pre-digested. Rest was absolutely insisted upon for many days after apparent recovery.

An epidemic of "colitis" has recently taken place in the Derby County Asylum, England. During twelve months ending last May, 54 persons were attacked and 23 died. "Nineteen necropsies were held, and the following facts ascertained: In five cases changes in the large intestine only were found; in four, changes in the small intestine; and in nine, changes in both large and small; in one case the stomach was ulcerated."¹

A specimen of ulcerated bowel was sent to the Jenner Institute of Preventive Medicine, and found to contain the "*Bacillus enteritidis sporogenes*."

One hundred and forty-four cases of hæmorrhage from the bowel are reported from St. Bartholomew's Hospital, from March, 1898. This epidemic was traced to the milk supply, and rods and spores of the same bacillus were demonstrated. "Three or four minutes boiling will kill the spores from an artificial culture, but six or ten minutes may be required to destroy them from the intestinal tract." It was

¹ Customs Medical Reports, lvii.

¹ Lancet, September 26, 1899.

noted in the Derby Asylum epidemic that the healthy inmates were less liable to be affected than the enfeebled or otherwise predisposed.

As regards treatment, bismuth, salol, opium, calomel, ipecacuanha, castor oil have been used here. In my opinion, calomel, castor oil, and chiefly ipecacuanha, are the only drugs which appear to be at all useful.

Washing the bowel by the insertion of a soft tube, morning and evening (preferably after a motion), appeared in my successful cases to diminish the number of stools and the hæmorrhage. I should have mentioned that among drugs salol appeared in one case to materially moderate the fetor of the evacuations.

Diet and absolute rest must again be insisted upon. Milk from the Pasteur Institute in Paris proved sweet and palatable, and the prepared foods (Mellin's and Benger's) suited the sufferers very well. Carelessness in diet was in my cases invariably followed by slight recurrence of hæmorrhage.

Recent Literature on Tropical Medicine.

TREATMENT OF THE PLAGUE.

The report of the International Commission on the Plague at Oporto, comprising such names as Calmette, Salimbeni and Ferran, is thus summarised in the *Revue Scientifique* (Oct. 21).

Experiments were made on animals with regard to (1) the establishment of passive immunity, and the cure of cases already infected by the injection of serum from the Pasteur Institute, and (2) the establishment of active immunity by inoculation with bacillary cultures prepared in the Ferran-Haffkine method.

They conclude that the serum is harmless, even in large doses (60 cc. daily), that injections of 5 cc. produce an immediate and efficacious, though probably not very lasting protection, and that the most promising treatment is that by large and repeated injections.

Active immunity, though more lasting, takes time (eight to twelve days) to develop, and during the interval the patient is perhaps less able to resist infection, for animals which were "vaccinated" and infected at the same time died more readily than those which received the virus only. This danger may be avoided, and the greatest possible protection obtained, by injecting the serum first, and vaccinating with heated bacillary culture about forty-eight hours afterwards. The Commission recommends that all persons in the immediate neighbourhood of an infected locality should be compulsorily treated either by injections of serum, or in the double method described above. In default of serum, the Haffkine vaccination should be carried out in two stages, a very small dose being followed in ten or twelve days by the normal quantity (about 2 cubic centimetres).

In this way they consider the spread of the disease may be always rapidly arrested, while the rigour of isolation might be considerably relaxed with regard

to all persons having a certificate of vaccination dating from more than twenty-eight hours, and less than a fortnight.—*Janus*, December, 1899.

Reviews.

HEALTH ABROAD. A Medical Handbook of Travel.

Edited by Edmund Hobhouse, M.D. Smith, Elder & Co., London. 1899. 372 pages. Price 6s.

The object of this book is to enable persons travelling abroad to preserve their health, and to supply information as to the treatment of climatic ailments until such time as medical advice can be obtained. Those who take the trouble to peruse the pages of this publication will, however find in addition useful information supplied them as to the art of travelling. They will be made acquainted with the clothing necessary to take, with the best routes available; what to avoid and what to attempt during a journey; in fact, they have placed before them in a concise form almost all that is necessary to know before starting and after they have reached their destination. Valuable information, truly, and in no published work can the intending traveller get information so reliable and so pregnant. The work has been apportioned to various writers, each one master of his subject, and the result is a volume of high merit.

Of the individual articles it is impossible to speak too highly. There is no attempt to puff, no writing up in a superlative form, the glories or beauties of this or that place. In fact, if we were to find fault, it might be said that every place has been written down, for with commendable candour the disadvantages, the drawbacks and the diseases of the several localities have been drawn with a pessimistic tendency, rather. This is as it should be. The gorgeous East is apt to fail to come up to the expectations of those who contemplate it from the advertisers' point of view, and it is well to have the facts of the case pointed out to intending residents and travellers.

Dr. Leigh Cannay, to whom has been entrusted "North Africa and Egypt," shows a grasp of the subject which does him infinite credit. Whether it is the little-known countries of Morocco or Tunis, or the more popular resorts of Egypt and Algiers, all are handled with a thoroughness that pronounces the work to be the handiwork of an expert.

South Africa is dealt with by Dr. B. J. Guillemard in a manner which shows an intimate acquaintance with the subject. Whether it is the climate and diseases of the littoral, the Karroo, or the high plateaus of the Transvaal, Dr. Guillemard is quite at home in his subject, and supplies information which the hunter, the soldier and the invalid, would do well to study.

Dr. C. F. Harford Battersby has been assigned a difficult and little-known region of Africa to deal with, namely, Central Africa. The writer of this section supplies us with a great deal of common-sense direction in regard to life in the tropics generally, and it will repay any intending traveller or resident in any part of the tropics to read his statements carefully and lay them well to heart.

North America and the West Indies are ably dia-

cussed by Dr. Edmund Hobhouse. Those who have travelled in Canada and the United States will readily appreciate all he says as to the facilities for travel afforded by the railway companies, and at the same time draws attention to the dangers of iced water, the canned vegetables and the fresh fish provided so liberally in the *menu* of American hotels. The *régime* followed in the hotels is ill-suited to invalids' wants, and the suffering that even the healthy traveller endures owing to the ventilation and heating methods inflicted upon passengers by the conductors is, unfortunately, only too true.

Dr. G. Herbert Pennell describes South America from a traveller's point of view, and states the advantages and drawbacks met with in a manner showing how familiar he is with the subject, which he approaches in a dispassionate and highly commendable way.

Australia and New Zealand are admirably handled also by Dr. Hobhouse. The writer presents us with a favourable opinion of Tasmania as a health resort, but warns invalids against going to Australia in search either of work or health, except they are strong enough to take to bush life. New Zealand has many advantages, and Fiji possesses the exceptional advantage of being free from malaria.

Travel in Europe, a difficult subject to handle, owing to its extent and intricacy, is described by Dr. Stuart Tidey in a manner at once useful and interesting. Switzerland naturally comes in for a large share of notice, and Dr. Tidey deals with the various localities, more or less well known, in a fashion which is highly instructive.

The Riviera and the South of Italy have special articles devoted to them, which are carefully written by Dr. Hobhouse.

India has been entrusted to Professor W. J. Simpson. A more able exposition of all the traveller and resident require to know concerning health in India has never been published. Concise, comprehensive and authoritative, the article will be highly appreciated and will prove a mine of information for Indian travellers.

The book ends with a chapter on the treatment of wounds and minor ailments, and a list of places where British practitioners reside.

We miss two subjects in this volume. There is no mention of travel in Japan and China. Japan, more especially, is visited by a very large number of Americans and English, and the omission of this country in the category of travel is a serious drawback to the book. The absence of maps, also, is a defect that ought to be remedied in succeeding issues.

We contemplate a rapid sale for this important work, and consider that "Health Abroad" ought to be a part of the *personnel* of every traveller.

THE MEDICAL ANNUAL FOR 1900. Wright, Bristol.

The appearance of this volume is ever welcome to the profession, and the present issue is even an improvement upon its excellent predecessors. To students of Tropical Medicine and to practitioners in the tropics the sections devoted to tropical diseases will be found especially useful. An article on Malarial Fever, by Major Ronald Ross, should be read by every

one who wishes to know the present position of this most important and interesting subject. Surgeon-Major Ross deals with the subject of malarial fever after an historical and critical fashion, and the new nomenclature employed in regard to the malarial parasite, &c., is fully explained.

Tropical diseases generally are dealt with by Mr. Cantlie. Since the "Year Book of Treatment" ceased to appear, we have no annual to compare with the "Medical Annual."

News and Notes.

ARTESIAN WELLS IN INDIA.—Looking generally at the map of India and its great drainage basins, there does not appear to be any probability of success attending artesian well-sinking in the country. No doubt water has been found in some small localities by deep borings; for instance, near Pondicherry; but in no great quantities of water so as to suffice for a largely affected area. I have very little doubt that further south, near Ramanad, similar wells might be sunk with success, as a strange sandstone formation runs all along that part of the east coast of India, so forming a basin in which artesian wells might be successful. But, for India generally, the Government must make up its mind to store water in large reservoirs, constructed on the largest scale possible. We have not been quite twenty years at work in Egypt, and the engineers there very quickly found out the absolute necessity of constructing a reservoir on such a river as the Nile, and the work has already been taken in hand. More than fifty or sixty years ago the late Sir Arthur Cotton proposed to adopt the same system on the rivers of India, notably on the Toongabadra, so as to supply most famine-stricken districts of the Madras Presidency with an abundant supply of water from a never-failing river.—*Indian Engineering*, February 10, 1900.

THE AGE OF MEDICAL GRADUATES IN JAPAN.—A Japanese paper, the *Jiji Shimpō*, finds reason for lamentation in the fact that whereas medical students in Europe graduate at 22 or 23 years of age, in Japan the age is over 26. The paper in question points out that the Japanese mature and age earlier than Europeans, and that as the period of usefulness in life in Japan is considered to be over at 50, the period of active practice is short. In Europe a man is usually at his best at 50, and even at 60, 70 and over, grave and reverent seniors are not looked upon as uselessly barring the way of ambitious juniors.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

DEAR SIRS,—With the approval of His Excellency the Governor of the Leeward Islands, I forward to you the enclosed Report on "Anchylostomiasis" in the Colony, as it occurred to me that it might answer very well for an article in your Journal.

Should you publish the paper, I would ask you to be good enough to put in a short editorial note drawing attention to

the fact that it is a Report made by me to the Government, who forwarded various documents, reports, &c., for my consideration.

I would take this opportunity of congratulating you on the result of your labours. THE JOURNAL OF TROPICAL MEDICINE is now an indispensable aid to medical practice in the Torrid Zone.

Dominica, W. I., Believe me, &c., &c.,
March 15, 1900. H. A. ALFORD NICHOLLS.

FOOD v. CANCER.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—Hippocrates, the father of Medicine, who died 361 B.C., taught that disease might be treated by medicine, by surgery and by diet. Medicine has had a fair trial, and has been in late years almost supplanted by surgery; but there are not wanting signs that the time is fast approaching when diet will be studied as it should be, and then even the surgeon will have comparatively little to do. Hitherto it has been a rare occurrence for a physician to call attention to the question of diet, and still rarer for a surgeon to do so; but now both physician and surgeon do not hesitate to enter the ranks of dietetic reformers. The last to join the pioneers of the profession is Sir William Banks, who, lecturing before the Medical Society on "Cancer of the Breast," says that as the result of his researches he concludes that overfeeding is the predisposing cause of cancer, and he attributes the increase of cancer to the more general consumption of butcher's meat, the use of which has increased enormously during the last thirty years. Physicians have long taught that gout was the direct result of overfeeding, and if their warning were listened to, what a fell disease would be banished from our midst. Some physicians have ventured to assert that overfeeding is also the predisposing cause of all our epidemic diseases, lowering the vitality and making us an easy prey to the various bacteria which give rise to such diseases. Now that a surgeon of such eminence as Sir William Banks, after a life study of cancer, with the accumulated wisdom of close observation, has come to the conclusion that the change from the simple food of our forefathers to the highly concentrated food, more especially the fleshy food of the present day, is the cause of that dreaded disease from which in the majority of cases even the unsparing knife of the surgeon cannot save us, may we not hope that the medical profession will in these latter days turn its attention to diet, and under the guidance of Hippocrates, who has left an excellent treatise on the subject, enter upon a crusade against all foods which are known, or can be proved to produce disease in man.

The laity have led the way so far; now let us hope that the Profession will take up the study of food, and that every medical school will endow a Chair of Dietetics, so that its students may be instructed in a subject hitherto entirely ignored by our medical educational institutions, and enabled to guide, not only themselves, but their *clientèle* in the choice of food, while in health, that all diet-produced disease may be stamped out.

I am,

Denholm, Yours faithfully,
Hawick, N.B., JOHN HADDON, M.A., M.D.
March, 1900.

Letters, Communications, &c., have been received from:—

- B.—Dr. A. Beveridge (Sidapur).
- C.—Dr. P. T. Carpenter (Punta Gorda); Lt.-Col. O. H. Chamer (Bombay).
- D.—Dr. A. Davidson (Edinburgh).
- G.—Dr. St. Geo. Gray (Castries).
- H.—Dr. Alice M. Hawker (Moorshedabad); Dr. W. C. Hossack (Calcutta).
- M.—Dr. A. J. M'Closky (Edinburgh); Miss E. L. Mitcheson, L.R.C.P. (Peshawar City).

- N.—Dr. H. A. Alford Nickolls, C.M.G. (Dominica); Dr. Chas. Neill (Barnet).
- P.—Dr. H. Campbell Perkins (Trivandrum).
- R.—Dr. E. S. Reynolds (Manchester).
- T.—Major C. R. Tyrrell, R.A.M.C. (Punjab).
- W.—Dr. W. de Wishart (Demerara).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
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- 6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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Original Communications.

THE ENDEMIC CENTRES OF PLAGUE.

By FRANK G. CLEMON, M.D., D.P.H.

III.—(Conclusion.)

In the preceding papers I have discussed at some length the prevalence of plague as an endemic disease in various parts of Asia, between the years 1850 and 1894. In the present paper the centres of plague in Africa will be dealt with in a similar manner.

The only places in Africa in which, so far as is known, plague appeared as an endemic or epidemic disease in the period in question are Assyr in Arabia, Benghazi in Tripoli, and on the shores of the Victoria Nyanza in Central Africa.

PLAGUE IN ASSYR.

The history of plague prevalence in Assyr is very far from complete. The earliest mention of the disease here relates to the year 1816, when it was believed to have been imported from Egypt by the Egyptian army. In 1853 plague was again epidemic in Assyr, and once more in 1874-79.

The table-land of Assyr is situated in the south-west of Arabia; it is bounded on the north by the province of Hedjaz (in which are the holy cities of Mecca and Medina), on the south by that of Yemen, on the east by the Nejd, or *hinterland* of Arabia, and on the west by the Red Sea. It lies approximately between the seventeenth and twentieth parallels of northern latitude. The part of the country adjoining the sea is flat, and is called the Tehama; farther inland the country rises, and the interior is mountainous. Living on the slopes or plateaux of these mountains

is a tribe known as the Beni-Sheir, and it is in this tribe that plague has from time to time prevailed. In 1874 the Beni-Sheir were described as "part of a tribe of 800 to 1,000 souls, spread over the Assyr mountains in encampments, distant ten or twelve days south from Mecca, six days south-east from Confuda and Leeth, and only three days from Mikhael."¹

Of the earlier outbreaks of plague in this region very little is known. Of that of 1874-79 there is on record a description, scanty it is true, but sufficiently detailed to establish, with something approaching to certainty, that the disease was in truth the plague. The region was visited at the time by Dr. Pasqua, of the Ottoman Health Department at Jeddah, and though he only saw five cases, he concluded from these and from the description of others that the outbreak was one of plague. The disease prevailed in many villages, and in some to so severe an extent as to carry off nearly one-third of the inhabitants. "Most of these villages," it is said, "are situated on the elevated table-land called 'Toumouna,' rising about 2,000 metres (6,500 feet) above the sea, and 1,500 metres (5,000 feet) above the 'Tehama,' and extending as far as the 'Namaz' range, from which it is separated by the valley of 'Waba.' . . . The outbreak prevailed at first on the plateau of 'Toumouna,' from whence it extended to that of 'Namaz,' and even went beyond it, as far as within four days' reach of Mecca. It then retraced its course and died out in the village of 'Namaz.'" The outbreak in 1874 began about the end of March, remained in a sporadic form until the middle of July,

¹ Report by Dr. Pasqua, of the Ottoman Health Department in Jeddah. See Parliamentary Papers. C. 2262.

raged violently through August, and then died down till it was nearly extinct in October. But it continued to prevail more or less in each of the following years, 1875-79. The disease was most active in Naumasse, the principal town of the Beni-Sheir, and in five other villages. The following information relates to this period of plague prevalence:—

"The site is on a mountain ridge too high for camels, the climate is cold and moist, the soil fruitful, springs abundant, and no standing water. The houses are built of stone and stand close together. The ground-floor of each house is used as the stable; and as the winter in these mountains is very severe, so that water freezes, the inhabitants live with their cattle in a horrible state of filth. According to the information from the district superintendent, there had been plague in a few villages every two or three years for the previous thirty-five or forty years. It has seldom extended further than five or six leagues. The region is a mountain canton with no trade, it is cut off from the rest of the world. The disease is mostly attended with buboes in the groins, armpits, and neck, but not always; sometimes petechial spots were spoken of; in the Sheikh Faik's own household the disease began with rigors, and developed buboes, petechiæ, headache and burning thirst. Dr. Nury counted up in six villages, with a population of 800, cases of plague to the number of 184 . . . with 155 deaths and 29 recoveries."

Two points stand out prominently in this description. One is the marked resemblance between the local conditions described here and those which obtain in Garhwal, Kumaon, Eastern Mongolia and the Persian table-land near Lake Urumiah, where plague has been either endemic or epidemic during the period now under review. The second important point is that plague had apparently been truly endemic in Assyria "for the previous thirty-five or forty years" before the year 1880, when the passage quoted was written; from this it must be inferred that plague had been there since 1840 or 1845, and that the outbreaks of 1853 and 1874-79 were epidemic manifestations of an endemic disorder.

It has further to be pointed out that, at the time of the revival of plague in Assyria in 1874, the disease was already epidemic in Mesopotamia, near the north-eastern borders of Arabia. That district of plague prevalence is some 800 or 1,000 miles removed from the centre of the disease in Assyria, and there was nothing to indicate that infection had been carried from one to the other.

It is interesting to note that a disease described as plague, or *waba*, is said to have ravaged villages in the northern part of the Nejd or Central Arabia, and lying in a direct line between Mesopotamia and Assyria, some years before the revival of plague in the latter region in 1874—probably about the year 1870. Doughty,² who, without visiting Assyria, journeyed through a considerable part of Arabia between November, 1876 and August, 1878, has described

a number of ruined and half-ruined villages which some years before his visit to them had been decimated by a fatal disorder. Of the villages in question the most important are those of Mogug, Gofar and Hâyil. They all lie near one another, approximately at the 28th degree of north latitude and 42nd degree of east longitude, and at a distance of some 600 miles from Assyria and some 200 or 300 miles from Mesopotamia. Of Mogug, this author wrote: "I wondered to see the village full of ruins and that many of their palms were dead and sere, till I learned that Mogug had been wasted by the plague a few years before." In Gofar the same condition was observed, and when an explanation was asked the answer was "*beled mât*" (a died-out place). "The villagers had perished, as those of Mogug, in a plague which came upon them seven years before" (consequently, about the year 1870). Wâsit and Hâyil were in the same state. "There died in Wâsit three hundred persons; in Hâyil 'one or two perished in every household (that were seven hundred or eight hundred), but now, the Lord be praised, the children were sprung up and nearly filled their rooms.'"

But, though this disease is spoken of throughout as "plague," it appears, from the brief description of the symptoms which follows, that the gravest doubts must attach to this view of its character. "Signs in the plague-struck," it is said, "were a black spot which appeared upon the nose, and a discolouring of the nails; the sufferings were nearly those of cholera. After the pest a malignant fever afflicted the country two years. . . . A townsman who brought down at that time some quinine from the north had dispensed ten or twelve grains to the sick at five *reals*; and, taken after a purging dose of magnesia, he told me, it commonly relieved them." From the entire absence of any mention of glandular enlargements or of severe pulmonary affection, it must be regarded as almost certain that the disease in question was not the plague. It would seem probable that it was either cholera¹ or some form of malarial fever, or the one succeeded by the other. It thus affords no link between the plague centres in Mesopotamia and in Assyria respectively.

Of the behaviour of plague in Assyria since 1879 little appears to be known. The only reference to the subject that I have come across was in the form of a brief statement in June, 1897, that the endemic area of plague in Assyria had been "free from cases for many months" before the outbreak in Jeddah in that year.³ Whether this implies that there had been cases some months before is uncertain. It was added at the same time that plague was believed never to have descended from the Assyrian mountains to the plains bordering the Red Sea, nor to the Nejd or Central Province. The caravans from Senaa (Yemen) and Assyria pass through the latter, but at some distance from the plague centre; they are inspected at Taif by a medical official, and no importation of the disease by this route, from Assyria to Jeddah, was detected.

¹ A. von Kremer; quoted by Creighton. "Hist. of Epid. in Britain." Vol. i., p. 166.

² "Travels in Arabia Deserta," by Charles M. Doughty. Camb. Univ. Press, 1887. Vol. i., pp. 578, 583, 617 and 618.

³ The year 1870, it may be noted, marked the commencement of a most violent cholera epidemic in Europe, which lasted for at least three years.

⁴ Note, from the late Dr. Dickson of Constantinople, to the *British Medical Journal*, June 26, 1897.

PLAGUE IN BENGHAZI.

The province of Benghazi, in the Regency of Tripoli, is the ancient maritime region of Barca, and includes the great plateau of the Cyrenaica or Pentapolis. A glance at the map of Africa will show that it occupies that prominence on the northern coast of the continent which projects, about its middle, into the Mediterranean Sea.

Plague is believed to have appeared here for the first time in 1858, in an Arab encampment of thirty huts, at a spot eight hours distant from the port of Benghazi, in the plain of Amalisgalen Fiddaar. In May it appeared in Benghazi itself, and then spread through four of the five divisions of the province, namely Benghazi, Derna, Gharb, and Chark, affecting both the nomadic and fixed populations. The outbreak began in April, 1858, and continued till the following year, finally dying out in June, 1859, after causing the deaths of some 4,000 persons.

The second outbreak occurred in 1874; it also began in April, but was of much shorter duration, and was over by July. It was limited to a few tribes of Arabs inhabiting the Cyrenaic plateau. The proportion of cases to the population was exceedingly high, as many as 533 out of a population of 734 contracting the disease; the case mortality was, however, remarkably low, both in this and in the earlier outbreak, being reckoned at only 39 or 40 per cent.

The outbreak in 1858 had been preceded by four years of drought, by a disastrous famine, and by great mortality among horned cattle, both from want of food and from an epizootic, the nature of which is not stated.¹ That of 1874 was also preceded by a severe famine, and the people at the time were in a state of extreme misery. "At the crisis of the misery, as it were, plague appeared in a diffusive form, and spread from encampment to encampment, and to certain of the villages and monasteries in the highlands, but not to the two towns of the littoral (Benghazi and Derna) or apparently to the tribes in the low-lying plains."²

The source of these two outbreaks in Benghazi was never made clear. Whether they were due to a truly endemic disease taking on epidemic characters, or to an importation of infection from elsewhere, there is nothing in the published accounts to show. It is at least noteworthy that the first outbreak (1858) occurred at a time when plague was known to exist as an endemic disease in Arabia; but before the re-appearance of plague in Persia (1863) and Mesopotamia (1867), from both of which countries the disease had been absent for some thirty years. The second outbreak in Benghazi (1874) coincided with the marked epidemic revival of plague in Arabia in that year, and with a prevalence of the disease in Mesopotamia (but not in Persia). All these countries are, however, far removed from Benghazi, and there was nothing apparently to show that the disease was carried from any of them to the latter. Nor is there any evidence to show that the infection was brought to Benghazi, as has been suggested by Prof. Koch, from the centre of plague in Central Africa.

PLAGUE IN CENTRAL AFRICA.

It has been known for some years that plague is truly endemic in the centre of Africa, close to the shores of the Victoria Nyanza Lake. How long the disease has existed here is quite uncertain.

The Rev. Robert Ashe, who lived for six years in eastern equatorial Africa, and whose book, "Two Kings of Uganda," was published in 1889, writes of this disease as no new thing. "This frightful malady [small-pox] and the plague, a kind of 'black death' called *kaumpuli*," he writes, "from time to time decimate the population of Uganda. *Kaumpuli* is recognised by the swellings in the armpit and in the groin, and by a terrible rise in the temperature of the patient. The fever runs its course in about twenty-four hours and usually proves fatal. Small-pox perhaps kills more people, yet it is not so much feared by the natives as *kaumpuli*, of which they have the greatest terror, as it is supposed to be frightfully infectious. Speaking of the *kaumpuli*," he continues, "I may refer to an incident recorded in the admirable book containing Emin Pasha's letters. Emin describes a conversation which he held with the Buganda guides who led him to Mutesa's capital. He asked them why Mwamba's land was totally depopulated. They replied that there was in Uganda a powerful magic called *kampodi*, no doubt this very '*kampoli*,' or '*kaumpuli*.' . . . The mistake which follows is, though very natural, a little amusing. Emin, believing the magic spoken of to be simply one of Mutesa's notorious raids, asked the guides if it affected goats and cows, or household utensils, 'and both gentlemen were silent.' If an intelligent Chinese were to ask us if the measles affected our silver spoons we should perhaps hardly be so polite as to keep a reserved silence. Emin draws the conclusion that Mutesa always enveloped his plundering expeditions in a veil of mystery."¹

Two years ago attention was specially called to this centre of plague by the observations of Prof. Koch and Dr. Zupitza. Prof. Koch was not himself able to visit the scene of the disease, but while in Africa he availed himself of the offer of Dr. Zupitza, who had recently come on leave from Bukoba to the coast, to go up country and make a full inquiry into the matter. He started on August 31, 1897, and it was only in February of the following year that his first report, accompanied by plague material, was received by Dr. Koch, who remained on the coast. In spite of all the difficulties of climate, mistrust and enmity of the natives, and lack of skilled help, Dr. Zupitza succeeded in collecting many clinical observations, in making four autopsies, and in obtaining preparations from the blood and tissues of plague patients. He also saw three instances of spontaneous plague in rats, and successfully induced the disease in rats, monkeys, and other animals. The specific character of the disease was thus proved beyond a doubt.

The natives, he states, call the pest *Rubwunga*. It has the same characteristics as plague in India. Rats die in enormous numbers at the same time as, or before, the outbreak in man; and to the natives of

¹ Parliamentary Paper, C. 2262.

² Ibid.

¹ "Two Kings of Uganda." By Robert P. Ashe, M.A., F.R.G.S. London, 1889. P. 130.

Kisiba this sign is so well known that as soon as rats begin to die the people forsake their huts. Both in Kisiba and Uganda the people live almost entirely on bananas; their villages are built in the midst of banana plantations, which are so dense that air and light can scarcely penetrate to them, and which swarm with rats—conditions which Koch rightly regarded as specially favourable to the multiplication of plague virus.

The exact position and boundaries of this area of plague prevalence seem to be very uncertain. Koch wrote (in 1898): "The real plague foyer lies beyond the borders of our [the German] colonies, on English soil. In Kisiba *Rubwunga* only appeared eight years ago, while in British East Africa, in Northern Uganda, it has prevailed from time immemorial. This I learn from well-informed missionaries who dwell in Uganda. These tell me that under King Mtesa—so well known through Stanley—the plague had caused terrible devastation in the capital of the country. Later it spread more to the southern part of Uganda, which is called Buddu, and from there was frequently imported into Kisiba. The circumstances under which it first appeared there are well known in all their details. It was, as already stated, eight years ago. At that time a man from Kisiba visited a business friend in Buddu. He there became infected, and died shortly after his return of *Rubwunga*. According to the national custom, his friends assembled to perform the funeral rites; they became infected and carried the disease into the neighbouring banana plantations, and so far and wide throughout the country."¹

Some of these statements have, however, been controverted. A paper upon the subject was communicated to the Literary and Philosophical Society of Manchester, in March, 1899, by the Right Rev. Bishop Hanlon, who has filled the Roman Catholic See of the Upper Nile since 1894, and is quite familiar with the disease and with the attitude of the natives towards it. His lordship disputes the statement that the infection had been brought from other parts of Uganda to Buddu, and thence southwards to German territory. On the contrary, he states that Buddu has for many years been a centre of plague in its worst form; and he believes that the disease was introduced into Uganda by way of German East African territory, which, he adds, has for many years been the chief route to that part of Africa. "It was *the* great slave route," he writes, "to and from the district of the Lakes. Speke and Grant travelled by it, Stanley travelled by it, the first Uganda missionaries came that way, and the French Fathers have ever used it. Our northern route is comparatively new."²

Whether brought from the north or from the south, or of local origin, the plague appears to have been most severe in Buddu. This is the south-eastern portion of Uganda, and lies on the west shores of the Victoria Nyanza and immediately to the south of the Equator. On German territory the places to suffer most have been Bukoba and Kitangule, on the shores of the lake. Bishop Hanlon stated last year

that he had heard from a German who had just come from the south of the lake that the plague was then confined to a small area in Bukoba. Finally, in July, 1899, an official announcement was made by the German Imperial Board of Health declaring that in the district round Bukoba and Kitangule plague was endemic. At the same time a statement was published to the effect that in Bukoba the Sultan had reported the occurrence of no less than 467 deaths from plague "in the past year," out of a population of only 715. If these figures are correct, they indicate an excessively high mortality. In the district round Kitangule, sixty deaths from plague were said to have occurred in the last four months of the year, 1898.¹

Whether this plague centre in the heart of Africa has any connection with that in Arabia, whether it had anything to do with the outbreaks of plague in Benghazi in 1858 and 1874, or with the supposed appearance of plague in Grand Bassam, on the French Ivory coast, in May, 1899, cannot at present be either asserted or denied. A like uncertainty surrounds the question whether this centre has shown any unusual degree of activity since the year 1894, when the disease began to spread far and wide from the centre in southern China.

CONCLUSION.

The review of the endemic centres of plague, as known to exist between the years 1850 and 1894, when the present so-called pandemic of the disease began its course, is now complete.

It will have been observed that these centres, while confined to Africa and Asia, and almost, but not quite, to the northern hemisphere, are nevertheless widely scattered. A mere enumeration of them is sufficient to demonstrate this: Mongolia, Southern China, the Himalayas, Mesopotamia, Persia, Arabia, Central Africa, and possibly Siberia (Transbaikalia), Russian Central Asia, and Tripoli. The endemicity of plague in Tibet is at present only a matter of conjecture.

A second important fact is that no connection has been definitely traced between any two of them, and no direct transmission of infection from one to the other has ever been proved, though it may have been surmised.

A third remarkable fact, or group of facts, is found in the variety of conditions met with in these different centres of plague. The disease has been no less truly endemic in Eastern Mongolia, where the winters are intensely severe, and the summers short and cold, than in the equatorial heats of Central Africa. It has prevailed at great heights in the Himalayas, and on the low-lying, alluvial plains of Mesopotamia; among races so diverse as the Chinese, Mongolians, Himalayan highlanders, Persians, Arabs, and full-blooded Central African negroes; and among the nomad tribes of Arabia and Tripoli as severely as among the settled population of some of the other places named. In short, there would seem to be no single characteristic or group of characteristics shared in alike by all the places and peoples enumerated—unless it be that all are somewhat removed from the great routes of trade

¹ *Hygienische Rundschau*, Berlin, July 15, 1898, p. 714.

² *Manchester Memoirs*. Vol. xliii. (1899). No. 6. "The Plague in Uganda," by the Right Rev. Bishop Hanlon.

¹ *British Medical Journal*. July 29, 1899.

and travel, and that in all the sanitary conditions and surroundings are at almost the lowest possible level.

Attention has already been drawn to the fact that in a certain number of these centres the conditions have presented many points in common. It would be easy to dwell upon the marked resemblances between the plague centres in Mongolia, the Himalayas, Arabia, Persia and perhaps Russian Central Asia—remote hill tracts, high above sea-level, inhabited by uneducated highland races, living in the midst of filth, poverty, overcrowding and other insanitary conditions. But in view of the behaviour of the disease elsewhere in the period under review and throughout historical times, it is obviously impossible to assert that these are the conditions necessary for the endemic prevalence of plague. In former years exactly the reverse view was held; low-lying, alluvial plains, and deltas at the mouths of rivers were regarded as the breeding-places of plague. Each of these alternative views is too narrow. Increasing knowledge of what is occurring in remote parts of the world, and a wider survey of the disease, tend to show that plague can become truly endemic under almost every variety of condition and circumstance. There is, perhaps, one important exception to this statement. It has yet to be shown that plague can become endemic in a town or country where sanitation and public health administration have reached a high level of general excellence.

It is almost superfluous to point out, after what has just been stated, that the "endemic centres of plague" are frequently changing. Were any other half-century taken than that now under review, other centres than those here described would have to be dealt with. Even the oldest of the centres mentioned in these papers dates back for no more than, perhaps, one or two centuries, and most of them are of much more recent date. Plague was for centuries truly endemic in Europe, yet it disappeared from it without apparent cause. Egypt was for long regarded as the home of plague, yet the disease completely left the country in an equally inexplicable manner. In a word, it would seem that there is no truly permanent home or breeding ground of plague, such as there would appear to be in the case of cholera.

Is it not possible that this difference in the behaviour of the two diseases is due to a difference in the life-history of the micro-organisms associated with them? That of cholera can, it is known, exist for long periods outside the human body, and it appears to find in many parts of India and the far East conditions favourable to its permanent existence as a saprophytic organism. That of plague, on the other hand, can, perhaps, only exist for very brief periods outside the bodies of human beings or the lower animals; and, as the conditions for the survival of an organism in the bodies of living creatures must be much less constant than those for the survival of an organism in a saprophytic state, so the organism of plague finds in no part of the earth's surface conditions for its permanent prevalence as an endemic disease.

Finally, I would repeat that a study of the recent history of plague shows conclusively that its wide epidemic spread since 1894 has not been the result of any universal or earth-change affecting simultaneously all those places where plague was endemic before that

year; but that it is in all probability to be traced to a diffusion of infection from one centre or group of centres only—those in southern China.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By GEORGE H. F. NUTTALL, M.D., Ph.D.

Pathological Laboratory, Cambridge.

(Continued from p. 233.)

Elevation in Relation to Malaria.—It is well known that only living in the upper stories of a house in a malarial district affords protection. Osler (*Pract. of Med.*, p. 142, New York, 1892) writes, "Persons dwelling in the upper stories, or in buildings elevated some distance above the ground, are exempt in a marked degree." Laveran and others make similar statements. As King puts it, malaria "hugs" or "loves the ground." Sleeping on the ground is particularly dangerous, because we are then more exposed to the bite of mosquitoes. Laveran cites, as examples of the effect of elevation, that at Constantine (Algeria) the mosquitoes are very numerous in the valley of the Rummel, which is malarious, but that they disappear as we go into the more elevated parts of the town, which are healthy. The same has been observed at Bone. Le Gendre (*Etude sur la topographie médicale du Médoc*, Paris, 1866, p. 26, cited by Hirsch) states that the zone of hills in the province of Médoc is only visited by malaria when the winds blow their way from the neighbouring swamps. Cornay (*Topogr. méd. de Rochefort*, Paris, 1845) and Crouigneau (*Réc. de mém. de méd. milit.*, vol. lxii.) both report similar observations in Rochefort and Rochelle. Russell (Address, New York Public Health Association, 1876, cited by King) states that under ordinary circumstances a certain altitude affords immunity, although low elevations of 200 to 300 feet above a miasmatic tract are often more dangerous than the flat lands, the poison seeming to float upward and become intensified. This, he adds, has long been noticed on the heights of Bergen Hill, West Hoboken and Weehawken, which overlook the Jersey flats. The mosquito, King remarks, "can readily be understood to be 'obstructed' and 'accumulated' by forests on the brows of hills, &c.," having been blown there by the wind. Koch (1898), writing of malaria in German East Africa, states that this disease is not found there at an elevation of over 1,200 metres, a point at which mosquitoes also disappear. It is needless to multiply examples, for the above observations have been made repeatedly.

The elevation at which malaria occurs is influenced by the average temperature prevailing at the place during the summer. This elevation naturally increases as we approach the equator. The freedom of the mountains from malaria is chiefly due to more perfect drainage. Where malaria does occur in the mountains it is always in valleys exhibiting but a slight fall, or in depressed imperfectly drained areas situated on the high plateaus. (Hirsch.)

(6) *The Role of Insects and Ticks in other Hæmatozoal*

Diseases.—Laveran (1896), Bignami (1896), Welch and Thayer (1897), and Koch (1898), have all brought out in support of the mosquito-malarial theory the evidence that ticks have been proved to reproduce Texas fever in cattle (see Texas fever), and that mosquitoes have been shown to be intermediary hosts to the *Filaria sanguinis hominis* (see Filariasis). The role of the dog-flea, in relation to *Filaria recondita* of the dog, is also suggestive. We have seen that young ticks may harbour and transmit the infectious agent of Texas fever to cattle, but the investigations of Grassi, Bignami and Bastianelli have conclusively proved that the malarial parasite is not transmitted to the young mosquito by its parent.

7. *The Coincidence of Malaria and Mosquitoes.*—Wherever we find malaria we find mosquitoes, but wherever we find mosquitoes we do not of necessity find malaria. As King puts it, all mosquitoes will not produce malaria any more than the scratch of every lancet will produce vaccinia, or the bite of every dog hydrophobia. The filarial diseases above-named, as also Texas fever, are also not found in all places where there are mosquitoes, fleas and ticks.¹ Lind (1757-1762) tells of an army half of which was lost whilst passing through Hungary, "The air swarmed with insects—a sure sign of its malignancy"; and referring to the climate of Guinea, the East and West Indies, as being fatal to Europeans, "more especially when molested with heat within-doors, and the plague of mosquitoes, they have ventured to sleep in the open night air." Laveran (1896) states that the French soldiers on the Madagascar expedition of 1895 suffered very much from malaria, and that they were "assaillis par des legions de moustiques." According to Manson (i. and ii., 1896) mosquitoes abound in Mauritius and Réunion, where malaria prevails. Ross (1898, ii.) made interesting observations in this connection in the very malarious Sigir Ghât or Cañon leading from the Ootacamund to the Mysore plateau. "Malaria commences three miles down the ghât, at a height of 5,500 feet above sea-level. At the bottom of the ghât 80 per cent. of the people examined had enlarged spleens, and mosquitoes were very plentiful at that point, being bred in a puddle lying within a few yards

of the bungalow and of an irrigation pool from which the servants drew their drinking water. Further down the ghât, however, and for its whole length, I did not succeed in finding a single mosquito grub in any of the pools of the ghât river or its tributaries, nor did I find a single one at Mr. Nash's plantation, where fever was prevalent." At first Ross was inclined to give up the mosquito hypothesis. "I was, however, saved from this conclusion by the discovery that, though larvæ could not be found in the ordinary sources of drinking water, the whole jungle abounded with fully developed mosquitoes to such an extent that it sufficed to sit down in the wood at noon to be surrounded by numbers of a virulent but small species of the insect," to which he gives the provisional name of *Culex silvestris*. This mosquito "appears to live entirely in jungle and undergrowth, especially in shady parts, and seldom, except perhaps at night, enters dwellings. Another remarkable difference in habits is that, while the ordinary species never travels far from pots and puddles containing stagnant water, the *silvestris* may, in my experience, be seen fully half a mile away from any water. Indeed I found it a matter of considerable difficulty to discover the larvæ at all . . . in a few scanty puddles at the bottom of nearly dried up and dark nullahs." One of his servants, who was employed in catching the adult mosquitoes "by allowing them to settle on his legs and arms, was attacked five days afterwards by the quartan parasite."

Joly (1898, pp. 44-50) considers that the evidence in favour of the transmission of malaria through the bites of infected mosquitoes is stronger or at least equal to that presented on behalf of the theory of its transmission by water.¹ He reports the case of a friend who spent two days in shooting in the marshes near the Etang Blanc near Tosse (Landes), where malaria prevails. The party to which he belonged took their food and drink with them, and avoided drinking any water in the infected district. Mosquitoes abounded, his friend was severely bitten by them, and eight days later, whilst in Paris, he developed typical malaria. He had never been in a malarious district before, nor had he ever previously had malaria. The infection through water being excluded, Joly considers that the mosquitoes are most probably to be regarded as having inoculated the disease. Joly also states in this connection that malaria is endemic on the borders of the pond of Cazau, several persons having acquired malaria there who came from Bordeaux. The water of the pond (*étang*) is used for drinking purposes. The downs in the surrounding forest contain many mosquitoes. Persons who traverse the downs or work in the adjoining woods are much troubled by these insects and nearly all who reside thereabouts acquire malaria. The water supply of Arcachon has been derived from the pond at Cazau for the last three years, but no malaria has occurred there. It is evident that if the water were the source of infection at Cazau it would be the same at Arcachon, but this is not the case. At Arcachon there are no marshes and mosquitoes are relatively rare. Joly considers that malaria may be

¹ Stebbins (1884), who criticises King's paper, considers that he has established its inaccuracy by the statement that he has been in various places where mosquitoes swarmed, but there was no malaria. Nicholas (1889) (I am indebted to the great kindness of Prof. Laveran for this extract) also considers the absence of malaria in the presence of mosquitoes is a ground against the acceptance of the theory. He writes: "En résumé, le moustique est souvent le compagnon du virus malarien, mais ce dernier peut se passer de son concours." To which Laveran remarks, in a letter to the writer: "En somme, Nicolas n'apporte aucun fait personnel à l'étude de la question." Hammond (1886) knows of no locality subject to malaria which is not infected by mosquitoes. Ziemann (1898) does not express himself in favour of the mosquito-malaria theory; he writes: "Kamerun, one of the worst fever centres on earth, is further but slightly affected with the plague of biting insects. (So there are mosquitoes there also. N.) During our stay on the western coast of Africa, I only once experienced a real mosquito plague; this was on the so-called Bimbria Creek, north of the Kamurun river, cases of malaria having already occurred before. It is known on the West African coast that every four or five years a particularly severe year for fever occurs. I never heard that in such years a marked mosquito plague was noted."

¹ All the experimental evidence hitherto gathered points very definitely against infection occurring through water.

communicated by mosquitoes which have acquired the infectious agent in malarial swamps, as also, but more rarely, by mosquitoes which have previously bitten a malarial subject. He also believes that infection may occur through water which may or may not have been infected by mosquitoes.

Koch (1898, i.) says that he has never seen malaria in places where there are no mosquitoes. He states that malaria does not occur on some small East African islands, for instance at Chole, which lies south of Mafia. "It was surely not an accident," Koch writes, "that this should be the only place on the coast where I found no mosquitoes and required no mosquito-net."

Dodd (1898) claims to have observed malaria in the absence of mosquitoes at Cæsarea in Asia Minor. This statement certainly requires confirmation from a competent source before it can be accepted, as it runs counter to that of all other certainly careful observers. (See further below.)

8. *Mode of Infection.*—Under natural conditions malarial infection must occur through the agency of air or water. There is no clearly positive evidence that malaria may be conveyed by water, and all experiments hitherto made have given negative results. Some observations (Laveran, 1898, p. 118), however, do suggest the possibility of this mode of infection. Manson, and also Laveran, believe that man may become infected by drinking water in which mosquitoes previously fed on malarial blood have died. They also believe that infection may occur through the inhalation of dust arising from dried pools which have harboured the parasite. This conception is based on Manson's observations in connection with *Filaria*, and of course the possibility of this mode of infection cannot, in the present state of our knowledge, be denied. A number of authors recommend the boiling of drinking-water as a prophylactic measure. On the other hand, King, Laveran (also), Bignami, Mendini, Koch and others, believe infection occurs through the bite of a mosquito; Manson believes that this is only exceptionally the case. The writer believes it is the rule in view of the evidence above given and that about to be presented below. The view that the mosquito carries the infection *directly* from man to man is untenable, according to (Laveran, Koch), for if this were the case the infection would be much more frequent. Though the disease is inoculable from man to man, and man to monkey subcutaneously, the quantity of blood inoculated has to be a considerable amount—far larger than it would be possible for mosquitoes to remove or reinoculate, supposing that they were capable (and that seems to me impossible) of inoculating by the simple introduction of an infected proboscis.¹

9. *The Malarial Parasite outside the Human Body.*—If we accept the mosquito-malaria theory at all, we are forced to the conclusion that the mosquito must be the *intermediary host* of the malarial parasite. If the insect gives rise to malaria through its bite, then

the parasite must be given off in the mosquito's salivary secretion when it is sucking blood. (I had come to this conclusion when the remarkable observation published by Ross, 1898, below referred to, came to give the supposition the much needed experimental support.) Knowing that malaria may be acquired in regions very rarely visited by man, we must regard the latter as but an occasional host of the parasite in nature. It remains then to be proved whether the parasite is capable of living an independent existence, or if it is always a parasite living on the mosquito or some other host besides man. Manson, Ross, Bignami and others, have expressed the opinion that the mosquito is the intermediary host of the malarial parasite of man. Bignami holds the opinion that the mosquito may become infected by the malarial parasite during its development in damp soil, and that the malarial germ may be primarily a parasite of the mosquito which by its bite causes malaria in man.

Both Manson (i. and iii.) and Laveran (1896) express the belief *that man may introduce malaria into a country by infecting the mosquitoes*, the disease becoming endemic. Lacaze (*Union Méd.*, 1872, No. 116, Hirsch, i., p. 211) states that malaria had existed for about three years in Mauritius when the first cases began to occur in the Island of Réunion. "Ici l'importation a eu lieu par Maurice, selon une probabilité qui touche à la certitude." Manson says mosquitoes abound in Mauritius and Réunion, and that now one-third of the deaths there are due to malaria.

ANCHYLOSTOMIASIS IN THE LEEWARD ISLANDS.

By H. A. ALFORD NICHOLLS, C.M.G., M.D.Abdn., F.L.S.

It would appear from Dr. Macdonald's report that anchylostomiasis was first recognised in Antigua by him early last May, and that since then he has had under treatment a number of hospital patients in whom the intestinal parasite has been found. He considers the presence of anchylostomiasis, which he describes as a "terrible disease," in Antigua to be "an alarming state of affairs"; and at the conclusion of his report he suggests that the worm is "quickly sapping the life and energy out of the labouring population of the island."

Dr. Macdonald has written an interesting report containing suggestions for the improvement of the sanitary conditions of the people, which, if given effect to, will do much good. I do not desire for a moment to say anything that might be considered to deprive him of the credit he is entitled to, but I think it is advisable for me to point out to the Government that there is no necessity for any undue alarm at the discovery of the parasite in Antigua, and that the term "terrible disease" as applied to those persons harbouring the worm is justifiable only in the case of a small proportion of affected persons.

The nematode worm known as *Dochmius duodenalis* (Leuckart), or *Anchylostomum duodenale* (Dubini), was first discovered in 1843 by Dubini in Milan; but it was not until an epidemic of fatal pernicious anæmia

¹ The experiments of Ross and of Grassi, cited below, have been published since the above was written, but they do not affect the above statement, for we see that the parasites must go through a *cycle of development* in certain species of Anopheles.

amongst the miners in the St. Gothard tunnel in 1880 was found to be due to the effects of multitudes of the parasites in the intestinal canal of the persons affected that the attention of medical men was directed to this newly discovered cause of disease. Since then the presence of the parasite has been demonstrated in many parts of the world, and in certain tropical and sub-tropical countries it is exceedingly common. Indeed, in India (according to Dobson and Rogers) and in Egypt (according to Bilharz) from 60 to 80 per cent. of the population harbour the parasite, and Matthias has shown that a very large proportion of the Europeans employed underground in the Kimberley and De Beers mines in South Africa are infested with anchylostoma.

In 1897 Galgey, of St. Lucia, asserted that anchylostomiasis is endemic and prevalent all over the West Indies, an assertion in which I entirely agree. I do not believe that the parasite has been newly introduced into these countries, for a study of the question can lead to no other conclusion than that many of the cases of cachexia Africana, or "mal d'estomac," so prevalent in slavery times, were due to the effects of anchylostoma. Indeed, the symptoms detailed by the early writers on West Indian diseases as characteristic of "negro cachexy" are identical with those set down in modern text books as diagnostic of anchylostomiasis.

The parasite is a small worm from 7 to 18 mm. in length which attaches itself to the mucous membrane of the upper part of the small intestine of man, and it acts in a manner similar to that of the leech applied to the skin; that is, it bites and then sucks the blood of its host. The adult female worms discharge innumerable ova which do not hatch out in the body, but require to be in contact for a time with the soil before the embryo is developed. The embryo then passes through several stages until the complete larval form of the worm is produced; and, unless the larvæ be introduced into the intestinal canal of man, they perish.

These facts show that infection can take place only by the transference of the larvæ from soil contaminated with human fæces to the digestive apparatus of man. It is evident, therefore, that these blood-sucking worms cannot be propagated except in those places in which the people—utterly regardless of the simplest rules of sanitation—are permitted to deposit their fæces on the surface of the soil. In most tropical countries this nasty and dangerous practice is the rule, and when the labouring population is dense, and the people are collected in villages devoid of latrines, the parasite is ever present, and cases of anchylostomiasis must be frequent. Ordinarily infection does not take place by water, and the larvæ cannot live in running streams; but, in those countries where pond water is used, as in Antigua, persons may be infected by using the water taken from the muddy margins. A pure supply of water, therefore, whilst preventing certain cases of the disease, cannot exert very much control on its prevalence as long as the soil is permitted to become constantly infected owing to the absence of latrines.

It is important to point out, in regard to the prevalence of the blood-sucking worm, that its presence

in any individual does not necessarily imply the train of serious and very often dangerous symptoms constituting the disease to which the term anchylostomiasis has been applied.

As each worm is minute, a very large number must be engaged in the abstraction of blood before a sufficient effect is made on the constitution to produce disease. A moderate abstraction of blood in plethoric individuals may not cause deterioration of health, inasmuch as the system is able to cope successfully with the loss. Dr. Leonard Rogers, a distinguished member of the Indian Medical Service, points out that 500 of the worms must be present for from six months to a year in order to produce anchylostomiasis or tropical anæmia, and he is supported in this view by other authorities. Except in cases complicated by disease, and in debilitated states of the constitution, the presence of even a few hundreds of the worms does not give rise to noticeable symptoms. Dr. Dobson, of the Indian Medical Service, shows that upwards of 80 per cent. of the healthy inhabitants of most parts of India harbour the parasite in numbers varying from units to hundreds. And Rogers makes the following important statement:—

"Dr. Dobson's observations, as well as those of McConnel, in Calcutta, and Bilharz, in Egypt, both of whom found this worm in the great majority of *post-mortems*, after death from accident or disease, prove that this worm may be present in comparatively small numbers in the majority of a population without doing any harm. I was able to confirm this in Assam, for I found this worm in 66 per cent. of fifty healthy men to whom I gave thymol. . . . Two hundred and ninety-three anchylostoma were passed by a man who had been in jail two and a-half months, yet he showed no clinical signs of anæmia. . . . It is evident that a very small amount of malarial fever exerts a much more deleterious effect on the blood than does a fair number of anchylostoma."

It is only within the last few years that the attention of medical men in the tropics has been directed to the question of anchylostomiasis, and it appears to me that, in the enthusiasm of working in a new field, many observers have over-estimated the harmfulness of what may be described as a moderate infection with the parasite. Facts that have come within my knowledge have satisfied me that the presence of a comparatively small number of anchylostoma has been set down as the cause of an anæmia due to the ravages of the malarial parasite in the blood. The fæces have been examined for evidences of anchylostoma, and the evidence to be obtained from the blood has been disregarded. In anchylostomiasis there is no enlargement of the spleen as in malaria, and yet, in clinical notes of supposed cases of the former disease, splenic enlargement (due to malarial infection) has been given as a prominent symptom.

I do not desire it to be considered from the above remarks that, in my opinion, anchylostomiasis is not a grave malady, but it appears to me to be necessary to direct attention to the fact that infection with *anchylostomum duodenale* is not anchylostomiasis. Anchylostomiasis is the disease due to the deterioration of vital organs by the drain of blood extending

over prolonged periods and produced by the bites of multitudes of the blood-sucking worm.

The parasite is not uncommon in Dominica, but owing to the sparse population and the abundant supply of pure running water in this large island, the chances of infection are much less than in Antigua. During the last few years a number of cases have come under my observation in which patients admitted to the Roseau Hospital have harboured the worm; but in only a few instances has it been possible to class the cases as pure ancylostomiasis. In one case I found the parasite in an European suffering from beri-beri, a disease which, I believe, has not yet been recorded as occurring in the Leeward Islands. In most of the other instances the parasite complicated cases of malarial anæmia.

An interesting case of ancylostomiasis came under my notice some years ago in private practice. A little girl, born in the West Indies of English parents, suffered severely from tropical anæmia (as it was called) and its attendant ills, and she had that peculiar symptom of ancylostomiasis known as geophagia or earth-eating. Unless watched, the child got out of the house and ate the earth with evident enjoyment. She was kept under careful and continuous observation, and treated with santonin and ferruginous tonics, and she completely recovered in a few months.

Since then thymol has come into general use in such cases; but, in order to kill the worms and cause their discharge, large doses of the drug have to be administered. Care should be taken, therefore, to determine the presence of very many ancylostoma before these somewhat dangerous doses are given, more especially in debilitated persons. Fortunately, with the aid of the microscope, the ova of the worm may be easily detected in the fæces, and their numbers in a single microscopic preparation will indicate approximately the quantity of adult worms harboured by the patient. The ova are recognised at once, and cannot be mistaken for the eggs of any other worm. It is, of course, not an agreeable task to make such an examination; but it is necessary, and its unpleasantness can be reduced to a minimum by the adoption of the following plan I employ at the Roseau Hospital:—A fragment of the stool to be examined is preserved by the nurse in a small shallow glass pot with a somewhat loose cover, and under the cover is placed a piece of loose blotting paper saturated with a solution of carbolic acid. The pot can be labelled with the name of the patient if several examinations have to be made; and, as soon as minute portions of a specimen are temporarily mounted for the microscope, the pot is filled with a carbolic solution to destroy all unpleasant odour.

The above facts concerning ancylostomiasis, and the life history of the worm, show:—

(1) That the worms, in their larval stages, are intimately connected with the soil.

(2) That the adult worms develop from larvæ after the larvæ are introduced into the digestive canal of man.

(3) That the worms act as minute leeches, extracting blood and causing little bleeding points by their bites.

(4) That owing to their minuteness, a multitude of the worms are necessary to produce any serious deterioration of the vital organs.

(5) That the worms cannot multiply within the body; and that, therefore, in most instances, fresh infections are necessary to produce disease.

(6) That as the larvæ are developed in the soil from eggs deposited thereon in human dejecta, certain obvious and simple sanitary measures are all that are necessary to arrest the propagation of the parasite, and, therefore, to rid any district of ancylostomiasis.

A NOTE ON MOSQUITO NETS AND MALARIA.

By R. W. FELKIN, M.D., &c.

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IN Dr. G. H. F. Nuttall's paper "Upon the part played by mosquitoes in the propagation of malaria," which appeared on p. 198 of the JOURNAL OF TROPICAL MEDICINE, I noticed with some interest that he, at p. 199, mentioned the fact that "Emin Pasha always took mosquito nets with him on his African journeys, considering that they kept off malaria."

It may perhaps be of some interest and perchance of some little use, if I briefly narrate what we thought on the subject and how we acted twenty years ago in Central Africa. For mosquito nets are not all alike; some are good and some are useless.

It is perfectly obvious that one cannot carry a mosquito-proof house with one on all journeys in what will be for many years an uncivilised country, however fast civilisation may spread. And even so, they could not always be at hand when wanted, even if one does admit, for the sake of argument, that the mosquitoes, which may and do convey malarial parasites, only bite at night, a fact I rather doubt.

But to the point. When in 1878 I was at Khartoum, on my way to Uganda, I remember well how one of the first pieces of advice Gordon Pasha gave me was never to sleep by any chance except in a mosquito net. That was not all, however, for he metaphorically stood over me whilst I personally had to make myself a mosquito net after his own model and to his own satisfaction, and he was not exactly easy to please.

He was convinced, he said, that the mosquito net acted as a filter against the malarial poison, as well as against the mosquitoes and other insects, which he thought might cause the fever.

The mosquito net was made as follows: and I may quote the words I wrote at the time and published in a book early in 1882. (Footnote—"Uganda and the Egyptian Soudan," vol. ii., p. 51.)

"I may mention how our mosquito curtains were made. The top, seven feet by three, is calico, the sides are mosquito netting, and at the bottom another piece of calico, the same size as the top, is sewn, except for about three feet on one side, which is left open as an entrance. Along the netting at this aperture we sewed a border of broad tape, filled with No. 2 shot. This acts as a weight and, as one creeps

in, it clings to the body and prevents the greatest plagues of Egypt accompanying us in too great numbers. When inside, a few mosquitoes are sure to be found, so that a boy holds a light at one corner of the curtain, towards which the mosquitoes fly and then are speedily dispatched. This plan is much better than having a light inside, for accidents frequently happen; and even if they do not, the light burns away some small fibres from the net and makes plenty of room for other mosquitoes to enter. To do Djour Ghattas justice (this was the place I was writing at) there were very few mosquitoes there; but it is a good plan for travellers in Africa to sleep always within curtains, as I have found this to be a great protection from malaria. The curtains should not be too dense on account of the heat, and also it is necessary to be able to see out of them distinctly."

I have still the original net made in Khartoum. The size was determined by the consideration that the angareb, which we used as a bed in preference to those supplied by outfitters, is that size, and, by the way, one great advantage of this angareb is that a porter easily carried one on his head, with the bedding in a waterproof roll, and it can be set down at once on a march and used as a seat, or if a mid-day siesta is taken one can sleep in the net.

The tip of the boy with the light is also worth noting by travellers. I should have mentioned in my description that he held his hand against the net, the mosquitoes flew to it, and the hand of the person inside then easily made an end of them. We always slept in loose socks.

But there was something more which I could not publish in a book for general readers, and that is this. Gordon Pasha made a special present to each of my companions and myself of a small iron enamelled cooking pot with a well-fitting lid, which was to be placed inside the mosquito net at night, to serve as a urinal, to prevent one needlessly exposing oneself to mosquitoes.

I do not think I ever slept out of my mosquito net for more than a week, and that was at Fatiko. I wrote then in my diary "Happily there were no mosquitoes." The result was curious; a short time afterwards I had my first bad attack of fever; this may have been only a coincidence, but it was a strange one and taught me very effectually the lesson not to sleep outside my net in the future, as it was a protection not to be dispensed with, even in the absence of mosquitoes.

Ever since I commenced lecturing on tropical diseases (in 1886) I have invariably impressed the great importance of the mosquito net upon my students. I hope that the few points connected with the subject which I have given above may be of use, as when travelling one cannot well trust to native servants and any arrangement which will ensure against carelessness is advantageous.

Emin Pasha and I had many long discussions on the part which mosquitoes and other insects might play in the production of malaria, but into this I need not enter now. I may say, however, that we found the castor-oil plant placed about the huts, and especially at the doors, a capital protection against mosquitoes.

WHAT BECOMES OF THE MOSQUITOES DURING THE DRY SEASON?

By ST. GEO. GRAY, M.B., B.Ch.

Colonial Assistant Surgeon, St. Lucia, W.I.

I HAVE tried to answer this question by investigating known breeding places of mosquitoes after the pools had dried up, with a view to discovering whether they had provided for the propagation of the species in the same place, or had migrated to a more congenial locality, to return to their old haunts when the rainy season recommenced and rendered them more favourable for the purpose.

On February 7, 1900, I examined a spot where I had obtained larvæ of *anopheles* a few months before. The pool had been dry for three weeks, hardly any rain having fallen during that time, and the surface of the mud at the bottom was cracked and dry, although soft enough under the crust. At 5 p.m. I took up some of this mud and a handful of grass from the sides of the pool and carried them home with me. I put the mud into a clean pickle bottle and the grass into another, and after putting about three inches of filtered water in each, I carefully covered them to prevent anything getting in or out, and awaited developments.

I had not long to wait. The following morning I discovered a few minute larvæ wriggling about in the bottle into which I had put the grass. These rapidly grew in size, and I soon had half-a-dozen healthy looking mosquito larvæ in my bottle. I was rather surprised to observe that these did not lie flat on the surface of the water like the other larvæ that I had obtained from the same pool, but behaved in all respects like ordinary *Culex* larvæ. On the 19th the first pupa was seen, and on the 21st, exactly a fortnight after I had taken the grass from the side of the pool, the first imago appeared. Within the next two days all my larvæ had become fully developed insects and turned out to be *Culex teniatus*, the commonest mosquito in this colony.

There was no result from the bottle in which I had put the mud.

I have tried several other pools in the same manner, but so far without result. However, I think that one successful experiment shows that some species of *Culex*, at any rate, do not always lay their eggs on the surface of the water, but where they will be washed into the pool by the first heavy rain.

I attribute my non-success in the other experiments to the fact that the grass had grown rapidly since the eggs had been deposited on it, and I should have taken it near the roots in order to obtain fruitful results. The task is not so easy as it appears, for an occasional shower will spoil all one's plans and the whole experiment will have to be begun all over again. (It is during dry weather and not when it is showery that these investigations are supposed to be made.) However, I think that there is no doubt that the eggs of *anopheles* remain fertile for a long time after drying, and I would suggest as a prophylactic, whenever practicable, burning the grass where *anopheles* breeding places are known to exist, so as to destroy the eggs before they mature, and thus prevent the pools becoming a source of danger to man.

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THE

Journal of Tropical Medicine

MAY, 1900.

THE LONDON SCHOOL OF TROPICAL MEDICINE AND ITS WORK.

THE London School of Tropical Medicine has been in existence scarcely nine months, and yet in that short time its promoters have every reason to be congratulated on its great and growing success. The objects of the establishment of the school were defined to be the acquainting of the students with the diseases of the tropics and their treatment, the investigating of tropical diseases, and the training to observe, record, and scientifically study tropical scourges. For these purposes, premises were specially built adjoining the hospital, containing well-equipped laboratories designed for about twenty students, and accommodation was provided for a small number (six) of resident students. Such, however, has been the

success of the school, that neither the accommodation in the laboratories nor in the residential part of the school is nearly sufficient, and it has recently been decided to increase both very considerably. By the proposed scheme the laboratory accommodation will be doubled, and not fewer than twenty students will be provided with residential quarters. The school has also been recognised as one of the thirteen medical institutions connected with the future University of London, and thus it has acquired at once a position of importance. The activity of the school is further shown in another direction. Under its auspices, and on the advice of Dr. Manson, the Secretary of State for the Colonies has decided to put to a practical test the mosquito theory of malaria. Dr. G. C. Low and Dr. Sambon, of the London School of Tropical Medicine, have consented to go out to Rome and live in the Campagna during the months of June, July, August, September and October, which are the most malarious in the year. A hut protected by wire netting has been constructed for them in England, so devised that it excludes mosquitoes. The entrance is by double doors, which are arranged to be self-closing and which are also protected by wire netting. Special attention is also paid to the ventilation, which allows of coolness and an adequate supply of air without permitting of the admission of mosquitoes. This hut is now on its way to Rome. It will be erected on a site chosen by Celli, who is giving his advice to the school authorities on this matter. The object will be to choose one of the most malarious sites. Dr. Low and Dr. Sambon will live and work in this hut. They will always have to be indoors by a certain hour in the late afternoon. The experiment will be watched with the keenest interest, for should it be successful, which we have little doubt it will be, unless some accidental or unforeseen defect occurs in the arrangements made to keep the hut free of mosquitoes, it will be the beginning of a new course in the preventive treatment of malaria, which is bound to have a very sensible effect in reducing the dangers of malaria-infected localities. The school, by undertaking this experiment, shows itself ready and able to engage in those scientific

investigations and studies which its promoters are of opinion are absolutely essential for the advance of tropical medicine. That scientific research in tropical disease must, sooner or later, be rewarded by results of the highest importance, receives most opportune support at the present time. The very remarkable discovery in the life history of the filaria made by Dr. G. C. Low, a student of the London Tropical School, and which we hope to publish in our next issue, is one which is destined to have a very important modifying influence on the prevailing ideas regarding the methods by which this disease is spread. Dr. Manson, in his original work on filariasis, discovered the part which the mosquito plays in the metamorphosis of the filaria, but he was unable to experimentally follow up the exact manner in which the filaria left the mosquito and entered the human body. He formed the view that the young filaria left the body of the mosquito when the latter died on the water, and that filariasis was a water-borne disease. Dr. G. C. Low has in the laboratory of the London School of Tropical Medicine almost worked out that part of the problem which Dr. Manson had to bridge over by an hypothesis. Dr. Low demonstrates, by a series of sections of mosquitoes filled with filaria by feeding on patients suffering from filariasis, that the filaria leaves the mosquito by way of the proboscis. It would thus appear that filariasis is, like malaria, an inoculable disease produced by infected mosquitoes—truly a most unexpected revelation. Doubtless the experiments will be carried further to test the accuracy of this inference, but there is no getting over the fact that the filaria escapes from the mosquito by way of the proboscis. Having seen the sections, we heartily congratulate Dr. Low on the excellence of his work, and the splendid results which have followed his close and prolonged investigation. Apart from the confirmation by analogy which the research affords to Ross's inoculation theory of malaria, it imparts an impetus to further investigation in the same direction, with the object of ascertaining whether the spread of other diseases may not be explained in a similar manner.

THE BRITISH GUIANA MEDICAL ANNUAL.

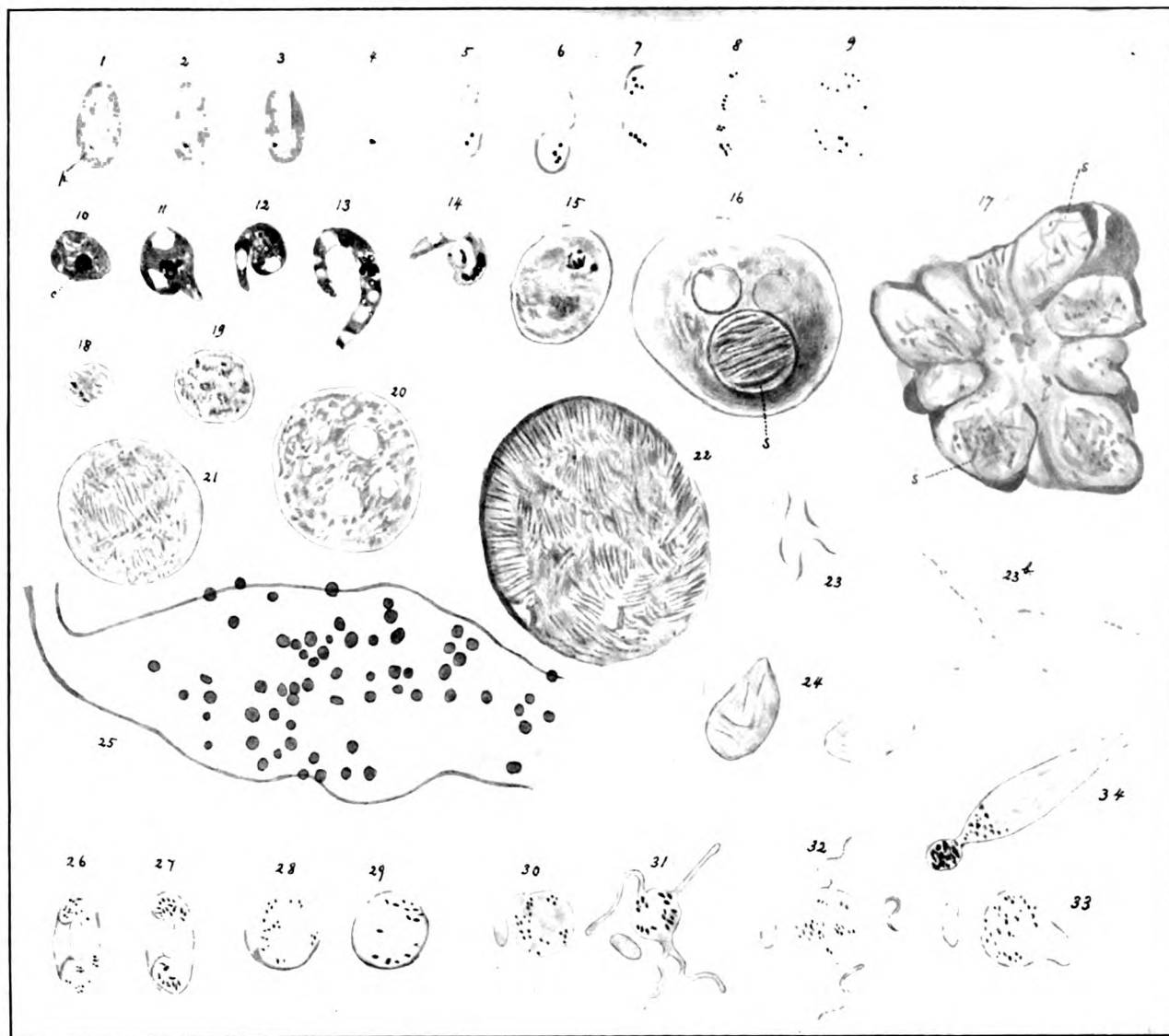
THE appearance of the eleventh number of this interesting Annual is not only welcome for the excellent articles it contains, but we would strongly commend the example set by the medical men in British Guiana, in publishing their experiences, to medical residents in other colonies.

The official reports required by the civil governors of colonies, like the blue books issued by the medical departments of the Navy and Army, contain, no doubt, useful information, but to the general medical reader they are so interspersed with administrative details that they cannot be said to be attractive.

When, however, a local medical journal is issued, the scientific work is separated from the mere administrative report, and the information supplied is more readily followed by the general reader.

Tropical diseases are met with over so wide an area; they are dealt with relatively by so few, and their number and importance are so great, that *every* contribution, even if confined to mere statements, is at once a help and essential factor in establishing on a scientific basis the geographical distribution and the nature of many of the tropical ailments. When the JOURNAL OF TROPICAL MEDICINE was started, it was announced that it was not intended as a rival to any journal extant; that it was not intended to supplant any periodical or annual; but rather, by gathering together information published in these journals, to serve as a medium of medical information between the different British and other Colonies. Did space allow we would publish in its entirety such a journal as the *Guiana Medical Annual*, and as it is, we re-issue a considerable part of it. We regret that the editor, Dr. Fowler, threatens, owing to lack of support, to cease the publication of the journal. We hope the threat will not be carried out, as it is in such publications more than in special articles to home journals that the local medical surroundings and the diseases incident to the place are spoken of. What is at hand daily, we but too often treat as

ILLUSTRATIONS ACCOMPANYING PROFESSOR G. N. F. NUTTALL'S PAPER, "UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A Historical and Critical Study."

DEVELOPMENT OF PROTEOSOMA IN *Culex fatigans* AND *C. nemorosus*.

- FIGS. 1-9.—Asexual development in red blood corpuscles of sparrow. 1-4 show the growth of the young parasite; the protoplasm increases in amount as also the chromatic substance. In figs. 5-8 the chromatin is splitting up into 2, 4, 8, 16 masses. Fig. 9 represents the young parasites being liberated from the degenerated corpuscles. $\times 1000$. (ad nat. G. H. F. N.)
- FIG. 10.—Zygote in blood twelve hours in *Culex nemorosus*.
- FIGS. 11-13.—Development of the vermicle.
- FIG. 14.—Vermicle about to cast off residual body. (Figs. 10-14 are after microphotographs by Zettnow illustrating Koch's publication in *Zeitschr. f. Hyg.*, xxxii. $\times 1000$. Romanowsky stain.) c. chromatin.
- FIG. 15.—Parasite in stomach-wall of *Culex nemorosus*. Second to third day. Unstained. $\times 1000$. Pigment is seen lying within a vesicle.
- FIG. 16.—Sporozoites formed within secondary capsules. On focussing down on this specimen the two smaller spherical bodies grow larger and are seen also to contain sporozoites. $\times 1000$. Unstained.
- FIG. 17.—Transverse section of veneno-salivary gland of *Culex nemorosus* containing sporozoites (s). In the middle lies the excretory duct. Stained with haematoxyline. (Figs. 15-17 are from microphotographs by Pfeiffer illustrating Koch's publication above cited.)
- FIGS. 18-22.—Progressive development of *Proteosoma* from the earliest encapsuled parasite (18) to forms containing sporozoites (21-22). Unstained. $\times 1000$.

FIG. 23.—Sporozoites. $\times 1000$. 23 b.—The same highly magnified.

FIG. 24.—Empty capsules. Unstained $\times 1000$.

FIG. 25.—Schematic view of stomach of *Culex* under low power. Stained and showing parasites dotting the stomach. (After original specimens and microphotographs of Ross.—G. H. F. N.)

HALTERIDIUM.

FIG. 26-34.—After McCullum, *Journ. of Experimental Medicine*, vol. iii., 1898.

FIG. 26.—Adult granular macrogamete in corpuscle.

FIG. 27.—Adult hyaline microgametocyte in corpuscle.

FIGS. 28-29.—Both of these (26-27) preparing for extrusion from the corpuscle.

FIG. 30.—Extruded macrogamete lying alongside of the nucleus of the red blood corpuscle.

FIG. 31.—Flagellation, i.e., microgametes being evolved.

FIG. 32.—Fertilisation taking place, a microgamete penetrating the female element.

FIG. 33.—The resulting zygote developing into a vermicle.

FIG. 34.—Motile vermicle with hyaline tip and pigment contained in a sack-like process at the posterior extremity. (The pigment-containing mass is thrown off, according to Koch, being composed of detritus which is of no further use to the parasite. See text.)

a matter of course; but what are common ailments in Guiana may be unknown in Ceylon; and the every day diseases of a place are often those of which but little notice is taken and less said. The earnest student of Tropical Medicine, the compiler of evidence as to the geographical distribution of diseases, the surgeon seeking for evidence of the nature and the results of surgery in tropical, as compared with temperate, climates, turn to the local journals, if there are any, for statistics and information. No such publication is without high value; each fills a niche in the history of medicine, which to those coming after us, or to ourselves in after years, will afford useful information, and will be the source from which reliable information will be gathered. We publish the several articles from the *British Guiana Medical Annual*, in the hope that other colonies will follow their example, and to assure them that the pages of the JOURNAL OF TROPICAL MEDICINE are ever open to distribute the information they contain to residents in the tropics generally.

MOSQUITO NET PROTECTION.

MANY persons aver that their immunity from malaria is due to the protection afforded by a mosquito net. These statements are in many instances so definite that they approach, if they do not even attain, scientific accuracy. We have only to turn to Dr. Felkin's article in the present number, to be convinced of the truth of this; but from many other sources we are continually being assured of the efficacy of the mosquito net.

One lady, relating her experiences during one and a-half years in the Canara districts of India, one of the most dreaded of the malarial regions of the Malabar Coast, states that she ascribes the immunity, enjoyed by her father, her maid and herself, from fever, whether in a tent in the jungle, or whilst living in a house, to the fact that all of the party on all occasions slept below mosquito nets.

Another lady, during a ten years' residence in

the East, always slept below a mosquito net except on one occasion. She had travelled in India, in China, Japan and Formosa, and it was only when caught unprepared on board a boat lying at the bar of a river in North Formosa, that she was minus a mosquito net. The solitary exception, however, proved the rule, for she contracted a sharp attack of fever immediately afterwards, which lasted a week.

Many Indian soldiers and sportsmen tell the same tale, and we have heard recently of the great care taken by the late General Whitlock, during his travels through India, to provide himself and those of his party with mosquito nets. One of the greatest modern travellers, Dr. Herbert Pratt, of Boston, U.S.A., is very decided upon the benefits of protecting himself from mosquitoes. For twenty-five years he has travelled continuously, and there is scarcely a state or province in the Eastern or Western hemisphere he has not visited. He never had fever during his experience of a quarter of a century of travel, and he ascribes his immunity from malaria and illness of any sort to always sleeping in flannel and being always completely protected from head to foot.

It is but the old custom of the Arab being brought to the knowledge of Europeans. The Arab, when sleeping out-of-doors, scoops a hollow for his body in the sand, and envelops himself completely in the folds of his ample garb. No mosquito can reach his head or his feet, for as he lies asleep no part of his body can be seen. Although all these travellers and tropical dwellers ascribed the benefits of the net to the warding off the "night-air"—the "miasma"—the fact of the immunity they enjoyed was none the less marked. We now know that it was not the "air" (that could not be kept out by an open net), but the barricade the net offered to the inroads of mosquitoes that accounted for the immunity.

ADDRESS TO DR. MANSON, C.M.G.—On May 14 the Students of the London School of Tropical Medicine presented Dr. Manson with an illuminated address on the occasion of his nomination as a Fellow of the Royal Society.

AN ENLIGHTENED POLICY.

THE Colonial Office has again shown that it is guided by far-seeing men, and by a statesman who has thoroughly grasped the question of Colonial welfare. The evidences of this fact have been frequently referred to during the last twelve months, and the most recent example is creditable alike to Mr. Chamberlain and his advisers. In the Federated Malay States, at Kuala Lumpor, Selangor, a pathological institute is about to be built and equipped on a scale commensurate with the importance of the contemplated work.

The laboratory is under the capable superintendence of Dr. Hamilton Wright, and whilst tropical diseases generally are to be dealt with, Dr. Wright's attention is to be specially directed to beri-beri. In the Malay peninsula, in Sumatra and Java, and in Borneo, beri-beri may be considered the disease which claims most victims, which hampers commercial undertakings, and delays the opening up of the country. North Borneo capitalists were ruined, and the industries of the country paralysed, some ten years ago, by the havoc beri-beri played amongst the Chinese coolies engaged on the estates, and many other countries tell the same tale.

Were the Pathological Institute at Kuala Lumpor to advance our knowledge of beri-beri alone, many a desert place could be made fruitful, and countries ignorant of tillage would be brought within the pale of the husbandman. We are proud to think that our country is waking up to the fact of its responsibilities in such matters, and that whilst its pioneers are extending its influence, the Colonial Office is alive to the only policy by which the tropics can be made to pay, namely by preventing the waste of life and by preserving the health of the British folk who take up residence in the tropics.

THE MOSQUITO AND FILARIAL INFECTION.

DR. G. C. Low, who has just been awarded the Cragg Scholarship in connection with the London School of Tropical Medicine, has, under Dr. Manson's guidance, added an important fact to

science. He has demonstrated in a microscopic section a filarial parasite in the proboscis of a mosquito. There is no gainsaying the fact, for the section is so perfectly clear that it is absolutely convincing.

At the Royal Society Conversazione on May 9, Dr. Manson, C.M.G., exhibited "Longitudinal Sections of Filariated Mosquitoes (*Culex ciliaris*), showing that *Filaria nocturna*, like the Malaria Parasite, leaves its mosquito host *via* the proboscis."

In the catalogue the following remarks were appended:—

"The insects were killed about three weeks after feeding on a girl in whose blood embryo filariæ abounded. In insects similarly fed and subsequently killed at serial intervals and sectioned, the passage of the filaria from the stomach to the thoracic muscles and their development there could be followed readily. The sections under the microscopes show that when development is completed, in about sixteen days, the filaria quits the thorax and becomes lodged in the head of the insect below the cephalic ganglia, whence it passes into the proboscis. It does not enter by the salivary duct, as is the case with the malaria parasite, but breaks through the base of the proboscis, passing along the upper surface of the labium between this organ and the hypo-pharynx. It is to be presumed that when the mosquito next attacks man the filaria enters by the wound made by the stilette."

Replies to Articles for Discussion.

ON THE RARITY AND ABSENCE OF SOME COMMON DISEASES OF TEMPERATE CLIMATES IN THE TROPICS.

ACUTE RHEUMATISM.

MAJOR BUCHANAN, I.M.S., on summing up, in the December issue of THE JOURNAL OF TROPICAL MEDICINE, an article by Dr. Banerjee, published in the September number of the *Indian Medical Gazette*, concludes by making the assertion, "Up to the moment we have no reliable statement that rheumatic fever (acute rheumatism) has been met with in the tropics."

Compared to its prevalence in temperate and cold climates there is no doubt that acute rheumatism is very rare in the tropics. I have met with typical cases, however, and on discussing the question recently with medical friends of wide experience, I have discovered that they all agree with me in pronouncing the disease as distinctly rare in India, though they have all met with undoubted cases.

In 1897, I had a very typical case: high fever with acid sweats; involvement of the knees, shoulders and one wrist; endocarditis; and, eventually, hyperpyrexia (the temperature rising to 110° F.) and death, as the parents would not consent to the employment of a cold bath, or even of a pack. The patient was a native child, aged 8 years, whose parents were in comfortable circumstances; and I have seen at least three other cases.

In the same issue of the Journal, Major Buchanan proceeds to dilate on the rarity of cases of organic diseases of the heart in India. He asserts that he has not seen a dozen cases in as many years, an experience totally different from that of various medical men with whom I have discussed the matter.

Personally, I have seen a large number of cases, and at present have two under treatment in the South Suburban Hospital, one of which is an excellent example of aortic regurgitation, in which the ventricle is beginning to yield. Last year I had two similar cases in hospital, and rejected several recruits for the Bengal Police for organic heart disease; and recently, with a view to throwing light on this point, I have examined the records at two of the Emigration Depôts in this town. Every would-be emigrant who is rejected for disease or disability, has his name entered in a register showing the actual cause of rejection; at one of the depôts I find that, out of 179 rejections, several were for "organic disease of the heart;" and at another, 45 out of a total of 237 rejections, the last working out to no fewer than 19 per cent. Those cases do not include individuals rejected for "anæmia with heart disease," "anæmia with heart murmur," or "weak heart." In my capacity of Medical

Inspector of Emigrants, I have the opportunity of seeing and examining every case that has been rejected by the Surgeon-Superintendent of the Depôt; hence I am in a position to be able to see and verify a large number of cases.

To continue, Major Buchanan remarks on the extreme rarity of cases of rheumatoid arthritis in India. I have always taken a great interest in this disease, and in the early years of my service was accustomed to make careful notes of every case observed, the result being that my case-books have a large number of undoubted instances, and I have recorded, among others, that of a girl of 10, it being very rare to see the disease at so early an age.

Again, as to the rarity of Chronic Bright's Disease in India. This is, admittedly, far rarer than in temperate climates, one reason being the extreme rarity of scarlet fever in the tropics, only a very few undoubted cases having occurred. Apart from its connection with this exanthem, however, there is no doubt that one sees cases of Bright's Disease in all its forms, and that they may occur without the slightest history of, or association with, malaria. I am afraid there is a growing tendency, in some quarters, to attribute to "malaria," a variety of disorders and symptoms, making that disease as responsible for abstruse and otherwise unexplainable phenomena as was gout not many years ago. It is an easy way out of a difficulty; but as our knowledge advances, I have no doubt that just as "suppressed gout" hardly finds a place in medical nomenclature, so we shall have less of "malarial dysentery," "malarial rheumatism" and "malarial eye-diseases."

Lastly, a few months ago, Major Buchanan remarked, in a paper contributed to the *Indian Medical Gazette*, that no case of appendicitis had been recorded as occurring in a native of India. I had seen three cases up to that time, and within the last four weeks have had the opportunity of observing three others. In one case, perforation of the appendix was the cause of general peritonitis and death; here a *post-mortem* examination was made for medico-legal purposes, and the fact was duly reported. The other two are in

my hospital at present; the one is extremely weak, though all the acute symptoms have passed, and I have deferred operation until the patient improves in his general health; I operated on the other last week and he is doing well.

From various medical friends whom I have been able to consult on the point, I have ascertained that appendicitis in natives of India is not a rare disease; and the fact that our experience differs so greatly from Major Buchanan's is doubtless due to the fact that his statistics are drawn, almost entirely, from the prisoners of the various jails of which he has been in charge; whereas ours is the outcome of the general population of large districts, and a very large out-door attendance of patients, as well as in-patients in hospitals.

E. HAROLD BROWN.

Major I.M.S.

Civil Surgeon, 24, Parganas.

Alipur, Calcutta,
February 4, 1900.

Reprints.

PREVENTION OF CONSUMPTION (TUBERCULOSIS) IN NEW SOUTH WALES.

THE following suggestions for prevention of Consumption (or Tuberculosis) are published for general information and guidance.

J. ASHBURTON THOMPSON,

President of the Board of Health.

Department of Public Health,
Sydney, 1899.

CONSUMPTION IS INFECTIOUS.

Every case of consumption is caused by infection received from some previous case. The previous case may have occurred in man or in one of the lower animals.

Infection from Man.—The infection spreads from consumptive man almost entirely by means of the phlegm thrown off the chest. Such phlegm is especially dangerous if allowed to get dry, for it can then break up into dust which floats on the air, and is thus likely to be breathed by the healthy. The phlegm infects everything on which it falls—handkerchiefs, books, papers, linen, carpets, floors, mats, roads, pavements.

Infection from Animals.—The infection can spread from animal to animal by the same means as from man to man, and also by milk. It spreads from consumptive animals to man when their flesh is eaten or their milk is drunk.

Disposition to become Consumptive.—People in good health usually resist the infection, but all can be rendered liable to take it by ill-health. The surest

cause of the habit of body which favours infection is living in badly ventilated rooms and houses. If in addition to being badly ventilated the dwelling be also dark, damp, and dirty, some of its inhabitants are still more likely to become consumptive sooner or later. The children of consumptive parents are often disposed to take the disease, though they are rarely born with the disease.

CONSUMPTION IS PREVENTABLE.

Consumption is preventable; for it is catching only in ways which can be easily prevented in part by the sufferer, and in part by the care of those living with him.

Destruction of Phlegm.—As the infection given off by consumptives lies almost entirely in the phlegm they spit up, this must be so spit out as to be completely gathered and afterwards destroyed. It must never be allowed to get dry between being spit out and being destroyed.

Cooking Milk and Meat.—It is difficult to be sure that meat or milk has come from healthy animals; but the infection of consumption, if it be in them, can be destroyed by cooking. Cook meat well. Never drink milk unless it has been first brought to boil. Above all, never give uncooked milk to children; they are especially liable to get consumption of the bowels from the milk of consumptive cows.

Fresh Air.—Ventilate the house freely by opening windows of living-rooms for a time each day. Never sleep with bedroom windows closed. Never block a chimney. Take care that the house is thoroughly well-lighted. Never continue to live in a damp house. It is better not to sleep on the ground floor unless the rooms are specially and thoroughly isolated from the soil by a layer of concrete or some more impervious material. Keep the house scrupulously clean. Remember that furniture requires washing as much as floors. Clean the walls.

Separation of Consumptives.—When the precautions described herein can be and are carefully taken, separation of the consumptive from his friends is unnecessary. But when the contrary is the case, separation is indispensable—from the unhealthy dwelling he occupies so that he himself may have a chance of recovering, and from the rest of the family so that they may not become infected also.

DIRECTIONS TO CONSUMPTIVES.

(1) Recovery from consumption is not uncommon. Perfectly fresh air, constantly renewed, is essential to recovery. Within doors never close windows entirely; avoid cold by wearing heavier clothing. Never sleep with bedroom windows closed. In this country it is possible to sleep out of doors during the greater part of the year; and this consumptives should do.

(2) The phlegm which is coughed up contains the infection. It must be spit out, and not swallowed, for it is infectious to the sufferer as well as to others. If swallowed it is likely to affect the bowels, which otherwise might escape the disease.

(3) The infectious phlegm is the chief means by which consumption is spread and kept going. It comes only from consumptive persons, and if they did not give it off consumption would not be as common as it is. As, however, consumptives do and must give

it off, all such persons owe this duty to their more fortunate neighbours—to so spit that the phlegm is safely collected, and afterwards completely destroyed.

(4) The best way of doing this is to keep a small jar handy; to line the jar with paper, folded so as to make a kind of open bag to spit into; to lift out the bag and the spit together, and burn them in the heart of a fire. Once a day the jar should be boiled in water for ten minutes or more, in a vessel kept for the purpose. Small jars with water-tight lids are made for the pocket, to be used out of doors.

(5) In-doors never spit on floors, mats, carpets, nor into fire-places. In public vehicles or public rooms do not spit on floors or mats, nor into spittoons for public use. Out of doors do not spit on roads or pavements. Take pains to spit into the pocket-jar, so that the phlegm can be destroyed at a convenient time.

(6) Carefully cover the mouth when coughing; do not speak near the face of another. The breath is not infectious, but the little specks of spittle given off when coughing and speaking are so. Do not kiss others, or only on the forehead. Keep hands and face scrupulously clean; pay special attention to the beard, &c., if one is worn. Sleep in a room alone if possible, but at all events alone in bed. Do not share food nor table utensils with others.

(7) Avoid using handkerchiefs as much as possible; substitute rags or soft Japanese paper for them, so that these may be burnt before they have time to get dry. Handkerchiefs, if used, and linen, if soiled with phlegm, should not be allowed to dry. They should be put in a vessel containing the disinfectant mentioned below, to soak for two hours at least before being sent to the wash. Do not have superfluous furniture in the room usually occupied, especially carpets, rugs, hangings, curtains, &c.

CLEANSING AND DISINFECTION.

Danger to the family can be entirely avoided by applying the foregoing rules steadily, and without causing the consumptive to feel himself at all excluded from his usual place in the household. But special cleansing of the room usually occupied by him, and of the house, together with disinfection of some articles, is necessary.

Disinfectants.—Sunlight and fresh air are excellent disinfectants, and they should be regularly and freely admitted to every part of the house; much can be done also towards disinfection of some articles which cannot easily be otherwise dealt with by exposing them for hours to sunlight and air outside the house. For all other purposes of disinfection connected with consumption the following should be used:—

Chloride of Lime	$\frac{1}{2}$ lb.
Water	1 gallon.

Stir well; may be used with or without the sediment.

The Room.—The bedroom and any other room usually occupied by a consumptive should be thoroughly cleansed at least once a fortnight. Wash the floor, skirting-boards, and furniture. Wipe every article, all ledges over doors, &c., and picture-frames very carefully with a cloth wetted with the disinfectant. Dry dusting must be given up; there is

danger in dust. Roll up rugs, pieces of carpet and the like, and spread them out of doors in the sun for a few hours; afterwards they may be beaten. Occasionally clean the walls of the room—if they are papered—by rubbing them with stale bread, carefully burning the crumbs afterwards; wash them if they are painted or varnished; colour-wash them anew if they are colour-washed. When a room ceases to be occupied by a consumptive it must be as thoroughly disinfected as after any other infectious disease. Full directions for doing this are contained in pamphlets on other infectious diseases, which may be procured gratis on application to the local authority of every district.

Linen.—Soiled linen may be disinfected in two ways. It can be boiled for ten minutes; but if inconvenient to wash it at once, or if it has become dry, it should be first cautiously steeped in a tub containing the disinfectant for two hours at least. Woollen articles may be soaked in the disinfectant.

Evacuations.—The evacuations of children and older persons who have consumption of the bowels should be received in a vessel containing a quart of the disinfectant, and should stand for two hours at least before being disposed of.

Scrupulous cleanliness in person and surroundings helps the consumptive to overcome his disease, and prevents those associated with him from taking harm.

Articles which appeared in the "British Guiana Medical Annual," 1899. Just issued.

SIMPLE DIRECTIONS AS TO THE TREATMENT OF MALARIAL FEVERS, &c.

By D. PALMER ROSS, C.M.G., M.D. EDIN., F.R.C.S. ED.
Surgeon General, British Guiana.

THE following simple directions for the treatment of malarial fever will be found very useful in tropical climates where the disease commonly prevails. In the hands of the writer during his period of service in Sierra Leone (1885 to 1894) the treatment was most successful, especially in what is now commonly known as "blackwater fever."

I.—MALARIAL FEVER.

(1) When there is headache, backache, shivering, lassitude, loss of appetite, nausea, and a tendency to constipation and slight fever (temperature 101°), give at once or at bedtime, one rhubarb and two colocynth and hyoscyamus pills, followed six hours after or next morning by a dose of white mixture with 10 grs. of quinine in it. If there is vomiting at onset of attack open bowels with castor oil, 1 oz., and a pint of warm water, as an enema. If the temperature is above normal and under 103° give fever mixture regularly as directed.

(2) When the temperature is high and over 103° give antipyrin mixture (or two 5-grain phenacetin tablets) every hour for three hours. This mixture reduces the temperature three or four degrees by profuse perspiration, in about four hours after the first dose.

(3) When this reduction takes place, give 20 grs.

of quinine in an ounce of water, and if there is a tendency to vomiting, administer it as an enema, mixing it with water only.

(4) About an hour after this, continue the treatment by giving a dose of fever mixture every four hours (10, 2 and 6 o'clock, day and night), until the temperature falls to 101°. If vomiting is persistent, give $\frac{1}{2}$ oz. of the mixture every hour.

(5) Now commence the quinine mixture, and repeat it every four hours for twenty-four hours, so long as the temperature keeps below 102°, or if necessary by enema as directed above.

(6) After this period should, there be no return of fever, give a daily dose of quinine at noon, and commence the arsenic mixture three times a day for a week, or until convalescence is thoroughly established.

(7) In cases where the urine is *black* or very *dark* coloured, give 20 grs. of quinine as soon as the temperature falls to 101°, and give a dose of arsenic mixture every four hours, so long as the temperature keeps below 102°. This mixture very soon changes the colour of the urine from black (like stout) to port wine, light claret, madeira, and lastly to its natural colour. During the time the arsenic mixture is being taken every four hours, it is necessary to give a dose of quinine mixture at 7 a.m. and at noon daily. If vomiting continues give instead a drop of Fowler's Solution in a teaspoonful of water every hour, say for twenty-four hours, and the quinine by enema. On cessation of vomiting resume arsenic mixture and quinine as before.

(8) Should there be a return of fever, resume the treatment as before, according to paragraphs 1 to 7, omitting purgative if unnecessary.

(9) If there is much vomiting during the course of the fever, apply a mustard plaster (4 × 4 inches) just below the breast-bone, and above the navel, for twenty minutes and continue treatment until the fever is controlled. Liquid blister applied behind each angle of the lower jaw, covering a space 1 inch by $\frac{1}{2}$ inch, is very useful. A drop of ipecacuanha wine in a teaspoonful of water every hour also relieves vomiting.

(10) If there is sleeplessness, give 30 grs. of bromide of potassium in an ounce of water at bed-time and repeat every four hours after if necessary. *Opium in any form is to be avoided except when absolutely necessary.*

(11) Stimulants—champagne or brandy—should not be resorted to unless there is nausea, urgent vomiting, or exhaustion, and then in a small quantity frequently, say a teaspoonful every quarter of an hour. Brandy to be diluted in an equal quantity of water.

(12) The diet should be plain and simple at first, and during the hot stage the following only should be taken: arrowroot or cornflour made with milk or water, chicken or beef tea, Liebig's extract, Bovril, Benger's Peptonised Beef Jelly, Brand's Essence, condensed milk (a table-spoonful to a bottle of soda water), and weak black tea. During the intervals of fever, give strong beef or chicken soup, made with a little barley or rice, with dry toast or plain biscuit. If the patient is weak, give eggs beaten up in milk, tea, or brandy and water. In cases of urgent vomiting give fluid nourishment in small quantities frequently, day and night, or by enema.

(13) All solid food should be carefully avoided until the temperature has fallen to normal (98.4 or 99°), and has remained so for forty-eight hours. Then give strong soup with dry bread or toast, lightly boiled eggs, tea, bread-and-butter, light puddings, custards, and gradually going on to fish, grilled chicken, mutton, beef, and vegetables, &c., &c.

(14) At the onset of a fever, the patient should go to bed and cover himself up with a blanket, and remain there until his temperature is normal, and not leave the house until he has been *free from fever* for at least forty-eight hours. Great care should be taken not to expose himself to a draught, as a chill followed by a return of high fever is sure to occur, and very likely end fatally. Full duty should not be resumed until convalescence is thoroughly established, and it is a safe plan to always bathe in tepid water, and especially for some time after an attack of fever.

N.B.—An attack of fever, however slight, should be treated with great caution, more especially after long residence without change to Europe, exposure in the bush, to rain, or the night air. Subnormal temperatures are dangerous in that they predict a sudden and high rise of temperature. Personally, I do not believe in acclimatisation in the West Coast of Africa, as the longer the residence the greater the liability to "black-water fever" and the worst types of fever.

(15) This treatment is useful also in yellow fever.

(16) Emetics should never be given at the onset of any of these fevers.

II.—DYSENTERY AND DIARRHŒA.

(17) When the calls to stool are frequent and the motions are soft, watery, and are accompanied with pain, give at once a table-spoonful of castor oil with 20 drops of tinct. opii in a small cup of strong coffee or with a little brandy.

(18) After the oil has acted and should the griping pains continue and the motions be scanty, contain blood or mucus, and have a slimy appearance, accompanied with fever, cutting pains round the navel, and a sensation of bearing or forcing downwards, give one pill of lead and opium, or 10 grs. of Dover's powder, every three or four hours, until the bowels cease acting. After this it is well to give one lead and opium pill night and morning, and a dose of quinine at noon for a week, and then one pill every day with the quinine for another week.

(19) The patient should go to bed and maintain as much as possible the recumbent posture. The pains in belly are relieved by the application of hot flannel wrung dry out of a pint of hot water mixed with half an ounce of turpentine, and continued every four hours for half an hour at a time. Twenty drops of laudanum in a little thin arrowroot or water, as an enema, will relieve the sensation of bearing down.

(20) The diet should consist of arrowroot or cornflour made with milk or water, weak black tea, chicken tea, Liebig's Extract, Bovril, raw eggs beaten up in milk or in a little brandy and water.

(21) Return to ordinary food must be very gradually made and great care and caution is to be exercised in what is eaten for two or three weeks after apparent recovery.

(22) Excess of alcoholic stimulants should be avoided, also immoderate quantities of fruit, especially

"Avacado pears," hot and highly-spiced dishes, shell-fish, oysters, English ale and stout.

(23) Care should be taken as to the quality of water used for drinking and cooking. For drinking purposes it should always be boiled, cooled, and then filtered.

III.—FORMULARY.

Antipyrin Mixture.

Antipyrin	grains	15
Carbolic acid and glycerine (1 in 20)	drops	20
Chloric ether	"	20
Water	ounce	1

Give this every hour for three hours when the temperature is over 103°.

In case of vomiting give 2 drachms every quarter of an hour for three hours.

N.B.—Should this medicine cause difficulty or shortness of breathing, this feeling will be overcome by a dose of brandy.

Fever Mixture.

Bicarbonate of potash	grains	10
Nitrate of potash	"	10
Chloric ether	drops	20
Carbolic acid and glycerine (1 in 20)	"	40
Water	ounce	1

Give every three or four hours during fever, and until temperature falls to 101°.

Quinine Mixture.

Sulphate of quinine...	grains	10
Water	ounce	1

Give when temperature is 101° and repeat every four hours, and so long as the temperature keeps below 102°.

Quinine Enema.

Sulphate of quinine...	grains	20
(Finely powdered).				
Water	ounce	1

Give this as an enema should the quinine mixture cause vomiting, and repeat every six hours as above (on no account add acid).

Arsenic Mixture.

Fowler's solution of arsenic	drops	3
Acetate of potash	grains	10
Water	ounce	1

Give this three times a day for a week after an attack of fever, or when free from fever, if the urine is black or dark-coloured.

MALARIA AND PREGNANCY.

By F. H. EDMONDS, M.R.C.S., L.R.C.P.
Georgetown, Demerara.

THE coincidence of malaria and pregnancy must always cause anxiety, for the foetal life is often sacrificed and the mother's endangered and sometimes lost. The subject divides itself into the effects of malaria—intermittent, remittent, and cachectic—on, first, the period from conception to the sixth month, and, secondly, from the sixth month to the end of the puerperal period. The simple intermittent has no effect on conception, and, if mild, no influence on the mother's or child's life.

In July, 1898, I saw a young girl who for the previous six or seven months had had a slight inter-

mittent, never going for more than a fortnight without chills and fever, which had been treated with quinine, arsenic, aconite, amongst other drugs prescribed by a dispenser. I was called in on account of persistent vomiting which had come on during the last two months, and, on examination, satisfied myself that the girl was probably at the end of the third month of pregnancy. Under treatment the vomiting—which I considered as the sickness of pregnancy—was stopped, and the pregnancy continued its natural course.

If, however, the intermittent is more acute, and the temperature rises over 104°, abortion will usually take place, as in the case of a girl who, having missed two menstrual periods, developed an acute intermittent (quotidian), and on the third day, with a temperature of 104·8°, passed a complete membrane containing a foetus before she had time to get the quinine mixture made up.

In all cases of pregnancy seized with acute intermittent the patient complains of pains in the abdomen, and the uterus can be felt hard and firmly contracted; but if quinine be given and the temperature does not rise to 104°, each succeeding paroxysm will have less effect on the uterus, and mother and child will be saved.

The (bilious) remittent malarial has the same effect on pregnancy as the simple intermittent, only differing in degree, the fever being usually higher; abortion is more common, and is often followed by a very large hæmorrhage of florid blood. Here also, if the case comes under treatment early, and the pregnancy be not taken into account, there is not much greater danger to the mother than in cases where there is no pregnancy. Malarial cachexia, notwithstanding the great depression and apparently low vitality of the subjects, is no bar to conception and does not interfere with the progress of pregnancy, nor need its treatment be neglected on account of the pregnancy.

Last year a young girl came to town from up the river Demerara. She was said to have had low fever every fourth day for nine months, and was, when I saw her, in a chronic malarial cachexia. I also found that she was about four months pregnant. After taking iron, bromides, and nux vomica she improved greatly, and returned to her home with every prospect of fulfilling her time successfully.

The absence of effect on pregnancy in these cachectics is probably due to the absence of high temperatures.

In the second period the complication of pregnancy with malaria gives rise to a much more acute and dangerous condition, due in great part to the reaction of the now partly independent foetus. In this period, if the woman be attacked by an intermittent during the first paroxysm, the foetal movements become very strong, and cause rather severe pain; they can be seen in many cases until the temperature passes 103°, when a painful, continued cramp of the uterus appears to limit the foetal movements; in this case the foetus may die, or it may live to react against another paroxysm; but in either case the mother will say she did not feel any more movement after such a certain paroxysm, although the next paroxysm will still be marked by the strong and painful contraction of the

uterus, and soon a paroxysm is accompanied by a miscarriage of a foetus bearing evidence of intra-uterine death of some few days, peeling epidermis and mottling, and the placenta is discharged of a heavy dull blue colour.

In October last year I saw a woman who was in her eighth month. At the time she was in the hot stage of the fifth paroxysm of an intermittent (tertian), temperature 104.6° ; the movement had been violent for the two first attacks, but had not been felt at the latter, nor during the intervals; the uterus was painful and very hard. Before she could get the prescription made up, she had miscarried, the foetus peeling, and the placenta gorged and leaden-coloured.

If the intermittent be not treated, the same painful, hard contraction of the uterus takes place at the succeeding paroxysms.

A bilious remittent malaria complicating the second period is the most serious condition we have to deal with; the reactions of the foetus are marked at first, but as the fever remits become weaker, they seem to fade away gradually, and not to cease after a certain definite effort. The contraction of the uterus is not so firm, but is painful, and there is much soreness and tenderness over the uterus and usually over all the abdomen. The adynamic character of this fever tends to cause delay in miscarriage or in parturition, and at the same time does not admit of the use of ergot, or of chloral, so that the process of delivery is very slow, lessening the chance of saving the child's life, which otherwise might be effected more frequently than in intermittent. However, if the temperature has risen much over 104° , the foetus will be dead before delivery.

When this bilious remittent appears during or just after parturition, the mother's life is placed in the greatest danger. I have seen young and healthy women pass to the last week of pregnancy in good condition, then fall off, become sallow-looking, owing to low remittent fever; during labour the temperature rises, the tongue gets thickly coated with a yellow fur, the patient becomes very restless, the pains weak and long drawn; after delivery there has usually been gushing of dark fluid blood; then an improvement for—usually—forty-eight hours, when a relapse (another paroxysm?) comes on with higher temperature, deeper jaundice, greater weakness and constipation, which, on relief being given by an enema, results in the passage of a large, black, stinking stool. After five or nine days' alternations—each marked by increasing weakness—the patient dies quietly, with many appearances of puerperal fever, but having had her lochia of good colour, quantity, and odour; and having had no uterine pain or tenderness. In these cases the child is frequently strong and healthy, but there is not a more dangerous condition for a woman than to be seized by a malarial remittent during her puerperium.

Malarial cachexia affects the second period of pregnancy much as any other condition of debility. The sufferer is reduced to a very low state by the cachexia, which is an asthenic state much increased by the disordered digestion usually present in these cases, where the stomach refuses to retain any medicine, and rejects even the lighter forms of nourishment. Labour

is very slow and lingering, with weak inefficient pains, and usually requires the assistance of forceps. The child is often born big and strong.

Twenty years' active work in malarious districts has convinced me that malarial fever is responsible for a large number of still-births. I am constantly seeing pregnant women suffering from untreated malaria, and always find that in the first paroxysm of intermittents, during the ague stage, the foetal movements are very strong and frequent, and the uterus not contracted. During the hot stage the uterus contracts more strongly, and continues contracting so much and so firmly that the foetal movements cannot be felt. I have always prescribed quinine, 5 grs., every four hours, and in cases where the temperature had not risen above 103° , and in which foetal movements could still be felt, have seen the malaria checked and the pregnancy proceed to term. I have also often seen, where the foetal movements had ceased after a previous paroxysm, and where the patient commenced taking quinine as soon as it was ordered, the miscarriage in a few hours of a foetus showing signs of intra-uterine death of some days before the quinine treatment was commenced. I have seen many cases where miscarriage has taken place, usually with evidence of intra-uterine death of many hours, in patients whose malaria had remained untreated. I have also known many cases of pregnant women during the earliest months taking 10 and 15 gr. doses of quinine and continuing the pregnancy to term.

I am strongly of opinion that malaria, by rapidly raising the body temperature, tends strongly to abortion and miscarriage, and also that the early use of quinine, by reducing temperature, lessens very considerably the still-birth rate.

DOES BERI-BERI EXIST UNDIAGNOSED IN BRITISH GUIANA?

By J. F. S. FOWLER, M.B.Aber.

I THINK I can safely say, if most of the medical practitioners of the Colony were asked the question, they would undoubtedly reply in the negative, although several clinical cases reported from here have, I understand, been diagnosed by a leading authority on tropical diseases as such.

That we have numerous cases of multiple neuritis there is no denying, and I feel sure, if they occurred either in an endemic or epidemic form, or in a country where beri-beri prevailed, and were attended by sudden deaths, would unquestionably be called beri-beri, and I feel equally certain that any practitioner accustomed to see beri-beri would on walking through our wards diagnose several cases they saw there as such. I shall therefore endeavour in this short paper to discuss the several types of cases resembling beri-beri that we get here, and the reasons for and against their being so diagnosed. That we have all the climatic and other conditions, such as overcrowding, &c., which are said to favour the disease, I think none will doubt; then it is owing to the absence of the germ (whatever that may be) that we do not get the disease developed.

That it is not epidemic or endemic, also, cannot be disputed, as all of the cases coming to the hospital

which present symptoms allied to the disease came from all parts of the Colony, no particular locality or estate being specially favoured. Now let us examine the type of cases.

(1) *Paraplegic Cases*.—Usual symptoms, partial and in a few cases almost complete paraplegia, certain amount of anæsthesia, varying very much in degree and situation, in different cases. In some, tenderness of the calves of the legs. Upper limbs, if affected, in a much less degree. General condition usually very fair. Muscles react well to the Faradic current, and this is never lost, but improves, that is to say, that the reaction to the Faradic current becomes more easily elicited, and with a weaker current, not like the reaction of degeneration. No ankle clonus. Knee reflex absent or very feeble. Peculiar ataxia, more a sort of falling forward movement, with marked inco-ordination. Ankle drop not marked. Usual way of trying to get about is by holding on the back of a chair and pushing the chair in front of them, and it is wonderful how well they get on with that aid. Micturition and defæcation give no bother. Digestion and appetite excellent.

Now let us look to the heart and circulation. In most cases there is nothing one can well diagnose as abnormal, in a few there is some slight dilatation of either of the ventricles, the action is usually weak, impulse feeble, but seldom if ever a bruit, and no noticeable change in the arteries, although the pulse may be a little weak.

(2) *Dropsical Cases*.—Numerous cases of dropsy will be seen in the wards, the amount varying from slight cedema of the legs to general anasarca, and at times ascites, but this latter is usually uncommon unless accompanied with advanced nephritis, or some affection of the liver. There is no marked disease of the heart, anyhow not sufficient to account for the symptoms; no albumen in the urine, liver apparently healthy, and blood in a fair condition. There is usually some anæmia, which varies greatly in amount, and commonly the ovæ of the *anchylostomum duodenale* in the stools, therefore most of these cases are diagnosed as *anchylostomiasis*, but I am of opinion that one is inclined to blame the poor *anchylostome* for too much; that its presence in any quantity is injurious to a high degree none will doubt, but on the other hand there are numerous far worse cases of *anchylostomiasis* going about outside, untreated and in apparently good health. The most remarkable point, I consider, is the rapid recovery these cases make. Given a good purge, thymol if A. D. ovæ are present, iron, suitable diet, rest and diuretics, they begin to improve at once.

Now, why are not at least some of these cases called *beri-beri*?

(1) The patients seldom, if ever, get sudden violent cardiac attacks.

(2) Sudden deaths among them practically never occur, most of the patients making a rapid recovery and leave the hospital very soon quite cured. It frequently occurs that a patient is brought to hospital quite unable to move from paralysis, marked dyspnoea and looking very ill, recovers and goes out in a very short time.

Why is this? Is it (a) on account of the change of residence, improved sanitary surroundings, free ven-

tilation, suitable dieting and rest in the recumbent position, or is it (b) that the disease is not *beri-beri*?

A colleague of mine (Dr. T. Ireland) to whom I mentioned these facts, is of opinion that it is certainly due to the first cause (a), as he informed me that he had in several cases in the country districts made *post-mortem* examinations in cases which had died suddenly, and he invariably found marked dilatation of the heart, with accumulation of blood in the right side, and serous effusion in the pericardium and cellular tissues.

Dr. W. S. Barnes, of the Public Hospital, Georgetown, is of opinion that most if not all the cases of multiple neuritis we get are due to alcohol, with which I must say I cannot agree, for the East Indian immigrants cannot be blamed for taking alcohol in excess, and the high price of liquor forbids the black man from indulging too freely; he may when he has the money go on a big spree, but they are usually few and far between, and not continued in the intervals.

There is another interesting point that should be considered. Lately, in most of the medical journals, a great deal has been written on the deficiency of fats in the food being the cause of *beri-beri*, which would certainly be in favour of our not having the disease among us, if such is the case, as practically all the black people and most of the East Indians, after they have been here a very short time, make use of fat salt pork or beef or other fatty substances at all their meals.

Remarks.—I am afraid I have given a very meagre description of the above facts, and must ask your indulgence for any omissions or shortcomings, as I have never had any personal knowledge of *beri-beri*, and so must leave the answer to you, as most of my opinions and arguments are based on Dr. Patrick Manson's description of the disease in his book on Tropical Diseases and other articles on the subject.

ANCHYLOSTOMIASIS IN BRITISH GUIANA.

By C. P. KENNARD, M.D., M.R.C.S.

THE occurrence of *anchylostomiasis* is so common in this Colony that there is a great tendency to put down all cases of anæmia to it, and I am inclined to think that on finding the A.D. ova we put down the anæmia to the *anchylostomes* and overlook other factors that may be causing it. In nearly every *post-mortem* more or less *anchylostomes* are found, no matter what was the cause of death.

On one of the healthiest estates in the Colony, out of two preparations of one stool in 49 cases, I found A.D. ova in 28. Of these 28 cases, 19 were in good health with no signs of anæmia; 9 showed some slight anæmia.

One of the 49 cases was markedly anæmic, and stools examined on three separate occasions showed no A.D. ova—she was a fever subject and drank only rain water from a vat; eventually she quite recovered from her anæmia. Another case was also examined three times, and although I could not find any ova, a few A.D. were passed after thymol; it is therefore very likely not to find them except by repeated examination, or not to have them with anæmia.

There is no doubt that there are acute cases of

anchylostomiasis, but I do not consider them of frequent occurrence. It is the chronic cases of anæmia in which the diagnosis of anchylostomiasis is to be questioned. It is clearly seen in the country that the general anæmia varies accordingly to the general healthiness. Some years ago, I was in a district in which for two or three years previously they had had excessive rain, and I was struck by the amount of anæmia, which I considered due to A.D.; three or four years after I returned to the same district and noticed that there was much less anæmia and the people generally looking better—in the interval the seasons had been drier, but otherwise there had been no change in their surroundings: generally speaking better water is drunk in the wet season than in the dry, although on the estates there is not much difference, which would point rather to the occurrence of anchylostomiasis in the dry season, whilst there is more sickness in the wet season.

The chief characteristics of the stools of the anæmic cases examined were the excess of mucus and the undigested food, the signs of chronic indigestion. This excess of mucus will prevent the dislodgement of the worms, more or less, which otherwise might occur in a normal intestine, and so an accumulation of the worms may lead to a direct result of their action; this catarrhal condition arising first and remaining being the principal evil. Catarrh of the intestine and thinness of portions of the intestines is a characteristic of the *post-mortem* in these cases.

That the thymol treatment removes some of the worms there is no doubt, but cases are often seen in hospital for months in which, after frequent doses of thymol and passing of worms, yet the eggs are still found and the general improvement is not marked; the majority of worms are probably expelled and the few that remain can do little harm—as they are so generally prevalent in healthy people there must be something else keeping up the anæmia; the indigestion and catarrhal condition of the intestines probably still remain.

I have found in some of these cases that the common worm-powder of calomel, santonin and quinine does more good than thymol. In a case I now call to mind in which A.D. were found and thymol was given, the man showed little improvement and had become a chronic; after some bilious vomiting I gave a worm powder and castor oil with the result in a few days of his being less anæmic and moving about the ward.

That malarial anæmia occurs frequently there is no doubt and the patients may pass anchylostomes. Pernicious anæmia I think occurs. In a case I had there was marked anæmia, slight general œdema and great tendency to sleep, A.D. were found and he had thymol treatment with no improvement. *Post-mortem* showed some few A.D.'s, organs healthy with the exception of some fatty degeneration of the liver and heart and some chronic bronchitis; as he was thought to have "sleeping sickness," the blood was examined before and after death for filaria and the worms searched for, but none found.

Incipient phthisis is sometimes taken for anchylostomiasis. Granular contracted kidney, when the process is very slow with loss of the urine and occasional albuminuria, is also I think a very common

cause of anæmia. In fact, any chronic disease with anæmia, if the anchylostomes are found, may be put down as anchylostomiasis, but the curing of the intestinal catarrh should be one chief object in the majority of anchylostome cases.

MALARIA: AS IT AFFECTS THE LOWER ANIMALS.

By J. BELL, Esq., M.R.C.V.S.
British Guiana.

In response to a request from the President to give the British Guiana branch of the British Medical Association a short paper on "Malaria as it Affects the Lower Animals," I do so, and in so doing I do not propose to go into the etiology of malaria, the causes of the pathological changes we observe in the blood and tissues, or its treatment—all of which you know far better than I do. I will merely give you the symptoms as I have noticed them in the horse, ass, mule, cattle, dog, cat and monkey.

Symptoms: Horse, Ass, Mule.—The two chief symptoms are progressive anæmia, and high temperature (say 105° F. with a variation of 2° or 3° either way) during the exacerbations, which occur at regular intervals of from two to seven days. The pulse is frequent and soft, the breathing quick and so liable to become distressed from slight exertion that such distress is often the first symptom that attracts the owner's attention. The mucous membranes become pale and yellow. The appetite is gone, condition is rapidly lost. The animal is dull and his movements languid, lazy, with "knuckling over" of the fetlocks. Pain is evinced on pressure being applied over the loins and on each side of the croup. The breath generally is offensive, especially in the cat and dog. Dropsical swellings take place in the extremities; a yellow, semi-gelatinous exudation is frequently found in the loose tissue under the skin.

Cattle.—In cows, together with the above symptoms more or less pronounced, we get suppression of milk, rumination is suspended, or carried on very irregularly. The muzzle or nose is dry and warm. That serene and placid expression to be seen on the countenance of cattle is gone, and tears are copiously shed. The horns are warmer than usual to the touch, and in passing the hand over the body a dry, harsh, hot feeling is very perceptible, different to that of health, which is smooth, oily and soft. The urine is secreted in fair quantity, but highly coloured. The stools are loose. The "knuckling over the fetlocks" is more pronounced than in the equine species, and the animal is inclined to adopt the recumbent rather than the standing position.

Dog and Cat.—The general symptoms observed in the equines and ruminants are also noticeable in the carnivora. The dog is listless, does not notice his master or mistress as before, will not obey commands as of old, he is lazy, he drags his hind limbs and appears tired and wearied. He seeks cool and dark places. The nose is hot and dry, the breath very offensive, gums and lips pale and anæmic—appetite bad. In the morning he appears better and livelier, but towards evening he gets languid and tired-looking. The exacerbations appear to be more frequent than in

other animals, and generally come on towards evening. Urine is scanty and highly coloured. Constipation is more or less present.

Monkeys.—I have seen what I suppose was malaria in four cases, inasmuch as they yielded in a short time to gr. i.-ii. doses twice daily of quinine. Their principal symptoms were loss of appetite and extreme lassitude. Instead of being lively and noticing one's every movement, they took no heed of what was passing, curled themselves up in a ball as if cold. Two of these patients had a bad reputation for biting strangers, but during their fever attack one could handle them with impunity.

Post-mortem Appearances.—Extreme emaciation, enlargement of liver and spleen; petechiæ on various internal organs, with a deposit of a yellow, jelly-like substance under the skin. The mucous membranes and other tissues are frequently tinged yellow with the colouring matter of bile.

I regret I have no fresh specimens of the blood to show; opportunities are plentiful, but circumstances under which cases are seen are not congenial to the obtaining and mounting of specimens.

In conclusion, I may state that the fever is very amenable to a brisk purgative of mag. sulph. and gr. ii. doses twice daily of quinine sulph.

Reviews.

BUBONIC PLAGUE. By Dr. José Verdes Montenegro, Professor at the Municipal Micrographical Laboratory, Madrid. Translated by W. Munro, M.D., late District Medical Officer, St. Kitts, W.I. (London: Baillière, Tindall & Cox.)

It is with much pleasure we have read Professor Montenegro's book on Plague. Ably written, it describes in a clear and brief manner the course, symptoms, means of prevention, and treatment of the disease. Beginning with the biological characters of plague, the bacillus is described, the methods by which it may be cultivated and stained and recognised, the influence of external conditions, the effect of disinfectants of different kinds, and the action on animals, are all dealt with and practical conclusions are drawn from them for preventive purposes. The mode in which the infection is manifested forms the subject of another chapter, in which infection through the skin, infection through the conjunctiva, infection through the respiratory organs, infection through the digestive tract, and infection through the vagina are discussed and given their relative places of importance. The vehicles which convey the infection, such as rats, fleas, man, and objects are mentioned, and the mechanism by which the plague is diffused touched on. Dr. Montenegro is inclined to favour the view that the fleas on infected rats are the agents in spreading the disease from these animals to man. "This mechanism of diffusion explains how at short distances plague is propagated with a certain regularity, spreading from one house to the neighbouring ones, from one district to another, seldom following the great pathways of communication from the interior of the cities or the most frequented roads between two neighbouring towns, but by mysterious

routes until the epidemic reaches the active period in which the number of those attacked is so enormous that it is impossible to establish the relation between the cases."

Of the gang of dustmen appointed to carry out disinfection in Bombay, only those became infected who lived in houses, as the poor do, of bad sanitary conditions; those who slept in the huts built by the Corporation, well constructed and without rats, remained healthy in spite of crowding.

With reference to the diffusion of the disease by rats, it is pointed out that a ship from an infected port may be the cause of an outbreak of the epidemic at a port visited by it without disembarking cargo, crew or passengers; the infection should not in such a case be ascribed to the air, as the rats can reach the land by cables or gangways.

Dr. Montenegro strongly advocates the modern régime of disinfection in place of the old system of sequestration. He would substitute medical inspection for the long quarantines, the lazarettos, the cordons, the closure of the frontier, and the suspension of all kinds of traffic, and he gives excellent reasons for the change.

Dr. Munro deserves praise for the interesting style which he employs in the translation. The book can be thoroughly recommended as an excellent *résumé* of the more modern views of plague. The volume consists only of some eighty pages, and the edition is inexpensive.

News and Notes.

LONDON SCHOOL OF TROPICAL MEDICINE, RESEARCH SCHOLARSHIP.—The £300 annual scholarship, announced by Sir Henry Burdett at the Festival held in connection with the establishment of the London School of Tropical Medicine, and now termed the Craggs Research Scholarship, has been bestowed upon Dr. G. C. Low. Dr. Low is a graduate in Arts of St. Andrews and in Medicine of Edinburgh University. He has just completed a three months' course at the Tropical School, where he has gained the approbation of the staff for his earnest work, his scientific methods and acumen, and his originality in research. The important discovery by Dr. Low of a filarial worm finding its way along the proboscis of a mosquito is mentioned elsewhere in this issue. On May 15 Dr. Low proceeds with Dr. Sambon to the Roman Campagna, to reside in a mosquito-protected house during the malaria season, and afterwards is to go to the West Indies to prosecute investigations there.

A WELL DESERVED HONOUR.—Amongst the fifteen new names nominated for election to the list of Fellows of the Royal Society, we rejoice to note that of Dr. Patrick Manson, C.M.G. Dr. Manson, during the past ten years, has been prominently before the scientific world for his work on malaria, but long before that date, whilst yet in an obscure corner of the far East, Dr. Manson, unaided by the advantages of modern research, had worked out the problem of mosquito-filarial infection, and established a scientific fact that has had a direct bearing upon his more recent

researches in malaria. Dr. Manson's work is so well known at the present moment that it is needless to recount it, and we are sure that all who know the valuable contributions he has made to science, in many departments of tropical medicine, are of opinion that no member of our profession has ever earned the honour more deservedly.

Correspondence.

To the Editors of "The Journal of Tropical Medicine."

SIRS,—I am indebted to the kindness of Mr. S. G. Hill, who has been recently engaged in medical mission work in Southern China, for pointing out a slight error in my paper upon the "Plague Centres in China" (in the March number of THE JOURNAL OF TROPICAL MEDICINE).

The error has consisted in regarding Lien-Chou and Lung-Chou (or Long-Tchéou) as one and the same place. Lien-Chou is near to Pakhoi, in the position described in the text of the paper referred to (p. 204, column 1, of the Journal). Lung-Chou, on the other hand, is at a considerable distance from Pakhoi, on the Tonkin frontier. Some of the outbreaks of plague mentioned in the paper occurred in one town, some in the other; but as, fortunately, the conclusions drawn from them are in no way affected by this correction, it is perhaps unnecessary to trespass further on your space and ascribe each year's outbreak to its proper town.

Mr. Hill points out that Lung-Chou (or Long-Tchéou) is of importance as being a customs station of the Chinese Government, and connected by railway with Tonkin. "Last year," he adds, "both Pakhoi and Lien-Chou suffered very severely from plague, and if the rule of former years will be followed, we ought to see a fresh outbreak at Hong-Kong this spring and summer."

I am, sir,
London, Yours obediently,
May 11, 1900. FRANK G. CLEMOW.

To the Editors of "The Journal of Tropical Medicine."

DEAR SIRS,—I am very interested in the article on "The Endemic Centres of Plague," by Dr. Clemow, in the current number of THE JOURNAL OF TROPICAL MEDICINE, having just arrived from Pakhoi, South China, where I have been for five years engaged in medical mission work at the Church Missionary Society's Hospital there. I write to call Dr. Clemow's attention to p. 204, paragraph 4 and footnote 8, where he confuses two important towns. Lien-Chou is not the same city as quoted by the French authors as Long-Tchéou (English orthography Lung-Chou). Lien-Chou is correctly spoken of as being about twelve miles from Pakhoi, but Lung-Chou is many miles away on the Tonkin frontier, and well known to the French. In the city near Pakhoi (Lien-Chou) there are no foreigners, and its name would not be known to any French outside Pakhoi.

Lung Chou (Long-Tchéou) is of importance as being a customs station of the Chinese Government, and being connected by railway with Tonkin.

Mr. Sharp Deane (whom I know personally) would refer to the city near Pakhoi in his Customs Reports. I think, if Dr. Clemow would consult *The Times Atlas*, he would see at once the two cities are different, though it would not affect his conclusions.

Last year both Pakhoi and Lien-Chou suffered very severely from plague, and if the rule of former years will be followed, we ought to see a fresh outbreak at Hong-Kong this spring and summer.

I do not write this for publication, of course, but (if you think the matter of sufficient interest) to be sent to Dr. Clemow, to whom I should be very pleased to answer any questions.

Yours faithfully,
Saxony House, S. G. HILL, M.R.C.S.
Cheltenham, March 28, 1900.

Letters, Communications, &c., have been received from:—

B.—Dr. Boddaert (Ghent); Dr. A. H. Browne (Umrissur); Mr. Benson (London); Dr. Fred. Burge (Shanghai).
C.—Miss A. Corthorn, M.D. (Karachi); Col. Cayley, I.M.S. (Weybridge); Dr. C. Christy (Bombay).
D.—Major R. Davis (Burma).
F.—Dr. Gundry Fox (Ipsch.); Dr. Robt. Felkin (Crouch End, London).
G.—Mr. A. Grumpelt (Barkly West); Dr. St. Geo. Gray (Castries); Major G. M. Giles (Naini Thal).
H.—Mr. L. G. Hill (Cheltenham).
M.—Dr. McDowell (Nigeria).
N.—Dr. George Nuttall (Cambridge).
P.—Surg. R. L. Price, R.N. (Hong-Kong); Capt. Pollock, R.A.M.C. (Paignton); Dr. Prout (Sierra Leone).
R.—Major R. Ross, I.M.S. (Liverpool).
S.—Staff-Surg. Shuttleworth (Deptford).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Esercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Mereck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

BLACKWATER FEVER.¹

AS IT IS SEEN IN BRITISH CENTRAL AFRICA.

By DAVID KERR CROSS, Blantyre.

HAVING been repeatedly questioned by the young physicians, now entering British Central Africa, as to the symptoms and treatment of this pernicious form of fever, and remembering also, that, ever since Koch's lecture on malaria at Berlin on June 9 last, specialists at home have given marked attention to the study of hæmoglobinuric fever, I have thought the present moment suitable for describing the disease from the point of view of one actually on the field who has seen and studied clinically many cases.

We will to a great extent leave theories and dwell on facts, adding our quatum to the ætiology of the disease.

It must be admitted that the actual cause of blackwater fever is still obscure. Some hold that it is the maximum of malarial infection, while others look upon it as a form of fever apart from ordinary malaria. Some regard quinine salts, given in extraordinarily large doses—120 grs. or more per diem—as a specific, whilst others, of equal authority, consider that quinine is prejudicial in the highest degree, even affirming that the outstanding feature of the fever—the black urine and extreme prostration—are signs of quinine poisoning.

It is only within recent years that blackwater fever has been differentiated and studied. It seems to

prevail in the most malarial districts of Tropical Africa and in milder forms in many other countries. Many medical men of repute in India affirm that it has only recently appeared there, and that it is on the increase.

Whether or not blackwater fever is malarial fever in an exaggerated form cannot yet be proved, but certain it is that while there are marked similarities—rigors, oscillations of temperature, perspiration, recurrence—there are also marked differences; we shall enumerate these.

(1) *Natives do not take this fever*—at least I have never heard of a genuine case—and they, to a limited extent, suffer from malarial fever of the tertian type.

(2) *Very many Europeans enjoy a complete immunity* from blackwater fever. The average of those who suffer, after careful observation, I would put down at about 10 per cent. Thus of 100 healthy Europeans, passing a medical examination at home, and coming out to British Central Africa, not more than 10 would take blackwater fever, while all would suffer from ordinary fever.

(3) *A European rarely takes this fever until he has been a year or two in the country*, and has suffered from a number of attacks of typical malarial fever. It is very unusual for it to attack the system during the first year of residence, and in my experience it most frequently appears during the *second* year. I can, however, recall one case—a fatal case—of the most virulent type, which occurred after only eleven months' residence. I should say that if the constitution has an idiosyncrasy towards blackwater fever, it will appear about the close of the second year of residence. If there is a predilection of the system, it will come on after, from eighteen months to three years. If the disease has not then manifested itself

¹ Circular for the Medical Department, British Central Africa Protectorate.

it is not likely to occur, and proves that the subject has in himself a special resisting power, or immunity against blackwater fever, and that his constitution has an adaptability towards the diseases of the African continent. This, of course, speaking generally. There is one notable case on record, of an African pioneer, who had this fever, of a pernicious type, for the first and only time, after twelve years' residence. He had always been subject to severe attacks of bilious remittent fever.

The above idiosyncrasies and adaptabilities run in families.—I can point to four different pairs of brothers, all of whom have suffered. In two cases one brother died from blackwater fever, and the other from the same cause was invalided home. In another case I know of, a father and child have had this fever, while the mother has not suffered. Again, there are instances of members of one family all taking fever after the same form. I can point to the case of three brothers and the infant child of one of them, all of whom died with an abnormally high temperature.

(4) *Those who are subject to blackwater fever may have ordinary fever*, say a regular intermittent, and yet have no tendency towards blackwater. I have at the present moment under my care a patient who had blackwater fever in February last, again now in July, and in September he has an ordinary remittent fever with apparently no blackwater symptoms. There is some as yet unknown difference in the two diseases.

(5) *No European who is subject to blackwater fever ever acquires an immunity from it*, while we all acquire in degree a form of immunity from ordinary fever. In the case of blackwater fever there seems to be the opposite, *id est*, a growing susceptibility to the disease. I think I express the universal experience in British Central Africa when I say that for the first year we all suffer greatly from forms of ordinary malarial fever—usually bilious remittent. In many cases these attacks come on once a month. After a year or two there is an illness of a more than ordinarily severe nature. Should this be overcome, a long period of fairly good health is entered upon. I do not affirm that fever will not even now attack the system, for at any moment even the oldest resident may be affected; but most of those who have been in British Central Africa for a length of time say that they do not now suffer as they did when first they entered the country. This immunity, I should say, is allied to that form of immunity acquired by the natives. They (the natives) have all doubtless suffered from fever, but their systems have acquired a resisting power. More striking still is the fact that those who have been, previous to entering British Central Africa, in malarial countries, enjoy comparatively good health from the very beginning. I know of two Europeans who have had such a record. One spent fourteen years in one of the most unhealthy parts of India, and another many years in a west Indian island, where malaria was notoriously prevalent.

(6) *Even after the sufferer from blackwater fever has left the infected or endemic area where he acquired the disease, both the ordinary and the*

blackwater form are apt to recur, and to retain their characteristic symptoms. This may occur within a year or more of departure, and points to likelihood of blackwater fever being due to some parasite peculiar to itself, lodged in the bone marrow, liver, spleen, or even the brain.

(7) *Unlike other fevers it appears to resist quinine.*—In fact quinine depresses the already overtaxed system. This drug is most defective in all forms where we have marked intermissions, less effective in remittent forms, and in my experience, harmful in hæmoglobinuric fever.

While blackwater fever usually begins like ordinary malarial fever it has always seemed to me that the initial rigor has been abnormally severe. Sometimes the sufferer is unable to take off his garments; he wrenches them off, being unable from the excessive shaking to undo the buttons.

The rigor continues, too, for an abnormally long time. Early uncontrollable vomiting and thirst set in. I have known of a case where vomiting continued every half hour, night and day, for three days, till death mercifully closed the scene. Thirst is always excessive. There is pain over the liver and spleen, but not more than in ordinary attacks. The temperature is usually not above 102° or 103° F., in fact seldom so high as in a tertian remittent. *The out-standing feature is the secretion of black urine.* Sometimes the sufferer is not aware of anything unusual until he sees the black or bloody urine. At times a large quantity is passed, at others only an ounce or two, notwithstanding the fact that he has a constant desire to micturate. Suppression of urine from the beginning is always a bad sign, and a free flow of urine, notwithstanding its colour, prognosticates well. The urine is only a *sign* of nature parting with the toxine secreted in the course of the blackwater fever. The fever continues of a remittent type, with, on some occasions, a severe prolonged diarrhoea, but speaking generally, constipation is present. After twenty-four hours or less, often, with a degree of suddenness, the whole body assumes a yellow tint. This icteric state remains, and even becomes deeper, as the fever continues. All the time the vomiting is distressing. There may be nothing in the stomach, but the retching continues, indicating that the toxin is irritating the vomiting centre in the medulla. What is parted with is usually bilious, although it does not seem to me to answer to the regular test for bile. There is something besides ordinary bilious matter present. In four cases I have observed, the urine gradually became more and more copious and clear; at first it was black and gummy, then a very dark brown, then it became like "port," then "sherry"; then gradually became natural in colour! In *five* cases the urine cleared up with a degrees of suddenness which caused me surprise; one hour the dark secretion was passed, and the next, a free supply of what appeared a normal secretion of the kidney. The urine may be completely suppressed, and this is a very serious symptom, but in such cases there never does seem to be the amount of uræmic poisoning one would expect; indeed, the twitching, &c., is absent. In one case there was suppression for fully forty-eight hours, and

when the catheter was passed, one ounce, of what appeared to be normal urine, was drawn off.

It seems to me that blackwater fever, as seen in British Central Africa, may be divided into the following varieties.

(1) *What may be termed paroxysmal form.*—I had, for ten days, to treat a typical case in a European, who had very marked quotidian fever. Every day about 9 a.m. he had a severe rigor with high temperature, at 11 a.m. he passed dark, bloody urine in considerable quantity. At about 4 p.m. we were astonished to find that the urine had suddenly assumed its normal colour, and continued so all night. Next day at 9 a.m., he went through the same paroxysms, with the passing of the same coloured urine, and the same clearing up. This continued for ten days, when he gradually got over his illness. Then I had a Sikh under my care, who had a marked quartan fever, with symptoms very much like the above, coming on every third day, only in this case, the dark urine continued to flow about thirty-six hours. I treated this fever with quinine and pig's bile, and he made a good recovery.

(2) *What may be termed a regular form.*—This patient has been in Africa for a year or more, and has suffered from ordinary fever, in the treatment of which he has used quinine freely, and is again laid low with fever. The rigor is more severe than usual, and he vomits incessantly. He has pains in his back and loins, and twinges of pain in the region of the liver. Three hours after the onset of the rigor he passes urine, dark in colour and thick in consistency. His skin becomes yellow, the fever does not abate even after several severe perspirations, and all the above symptoms continue—vomiting, pain, &c. On the third day, in some cases gradually, the urine passes through shades of colour till it reaches the normal tint. The yellowness of the skin passes off gradually. When the fever leaves the patient he is as weak as an infant. The crisis is reached on the third day. The temperature oscillates a good deal from 99° to 105° F. Death in some cases occurs from hyperpyrexia, but this is rare. Very often on the appearance of the normal secretion the patient makes a good recovery.

(3) *What may be termed a typhoid form.*—The first three days of fever are exactly like the regular form. There is a severe rigor, severe vomiting, marked icterus, dark-coloured urine, and on the third day the urine clears. There is great hope of a speedy recovery. The attention is taken from the bloody-coloured urine to other symptoms. The temperature remains abnormal and oscillates. The brain is markedly affected, as is shown by the low muttering, the tossing of the body from side to side, and hallucinations. The patient may not sleep for days. The tongue is dry and parched. There is very often uncontrollable diarrhoea. Often hiccough, which cannot be controlled, tears the unhappy sufferer. This may continue for a week or ten days, and often leads to death from exhaustion. There were two deaths in British Central Africa from this form during the present year.

(4) *What may be termed a suppressionary form.*—Here the urine becomes more and more scanty, until

it ceases altogether, or what is passed is of a gummy consistency, and can be drawn away in long dark shreds. It is loaded with albumen. The case usually terminates on the third day in convulsions or coma. In one such case after long suppression the catheter was passed, and a tablespoonful of what appeared to be normal urine was drawn off.

Complete suppression from the beginning, or a gradually lessening and darkening secretion, prognosticates a serious end. On the other hand, a suddenly developed darkness, with urine secreted in abundance and tending to clear up at an early stage, always forebodes a good recovery. The most pernicious cases usually end in death. The fever may assume the typhoid form wherein the uræmic symptoms are not so marked, but the brain is seriously affected, when death takes place with an extremely abnormal temperature. At other times the patient has all the symptoms of severe hæmorrhage, profuse perspirations, faintness, sighing, and tossing of the body from side to side. In rarer cases the kidneys may be so affected by the toxine, that weeks after, death takes place with signs of inflammation of the kidney.

There are three physical signs that are marked in Blackwater fever.—Two of these are apparent to the casual observer, the third can only be seen by the physician. (1) *The black or blood-red urine*; (2) *The development of a yellow colouring matter* which invades every tissue in the body; and (3) *An abnormal black colouring matter—melanin*—floating in the blood plasma, or inside the affected red blood corpuscles.

The characteristic black or red urine gives the popular name to the fever. A few ounces of this urine, like prune juice in colour and consistency, may be passed daily. It is usually thick with albumen, becoming solid when boiled like pure serum, and apparently loaded with blood, but at the same time it has not one blood corpuscle. If this urine is allowed to settle it quickly manifests three well-marked layers. The upper layer is usually in colour like port wine, but transparent. The second is a thin layer, of a darker tint, but cloudy, as if ordinary mucus were suspended in it. A lower, and a third layer is apparent, which is very dark, non-transparent, and looks solid or flaky. This sediment consists microscopically almost wholly of disintegrated material, which seems to be amorphous. Clear and red tubercasts may be seen at times, which are apparently due to the colouring matter of the blood-hæmoglobin. We are told that the spectrum of the hæmoglobin is in some way altered. A great deposit of reddish amorphous material is seen, which I take to be urea. On looking carefully, the epithelial lining of the tubules of the kidney are seen, but blood corpuscles are conspicuous by their absence, although we all look for them with care. It is not a hæmorrhage but a hæmoglobinurinae.

In every Blackwater fever case there is an enormous deposit of another substance—a yellow colouring matter. The patient suddenly becomes yellow all over. The whites of the eyes become as yellow as ochre—a guinea yellow. This "pigment ochre" as Kelsch calls it, is a most important factor in the case. It is said to be found in every disease in which the destruction of the colouring matter of the blood is serious

It is found in pernicious anæmia, and in death from drugs such as chlorate of potash, glycerine arseniate of hydrogen, in those cases where quinine cannot be borne, and in many other instances. This yellow substance is the product of the destruction of the R.B.C. themselves. In ordinary circumstances the liver converts this substance into bile-salts which pass off through the usual channels. When, however, the destruction is excessive, it would seem either that the liver is unable to cope with the abnormality, or owing to the necessity of the case the other tissues take on the functions of the liver and form bile. Bilious symptoms are seen more or less in every form of malarial fever, but they are marked in bilious, remittent and blackwater fever. Bile is poured into the stomach and vomited into the bowels and parted with. Some hold that the yellow ochre so manifest in the tissues of the body, is due to the presence of free hæmoglobin in the blood and tissues.

The liver is doing its utmost, as it were, to work it off, and the stomach, bowels, kidneys, and *skin even* come to its assistance and part with what appears to be injurious. I very much doubt, however, if what is taken for bile is really this necessary substance. It does not appear to me to react to the proper test. Perhaps, after all, what the burdened system needs is real bile.

There is a third abnormal colouring matter present in this fever which stamps it more and more as of the true malarial type. I refer to the black pigment of the blood—melanin. This substance is always seen in the life history of the Plasmodium malarii. In some stages it is inside the red blood corpuscles which are affected by the parasite, and each variety of parasite seems to manufacture a special brand either as amorphous dust, or rods or clumps. At another stage it is free in the plasma of the blood or inside the bodies of the phagocytes. This melanin is the product of the digestion of the hæmoglobin of the R.B.C. by the malarial parasites.

*It is an interesting question as to what the above-mentioned yellow colouring matter really is!—The melanin we know! What is the yellow colouring matter? Specialists tell us, and we can see for ourselves, that the R.B.C. which remain in the blood of a subject of blackwater fever are not normal. They are not actually invaded by the parasite as far as we can see but they are lighter in colour than normal. In some cases they appear smaller than normal, and in other cases they appear larger, and when placed on the slide manifest a disinclination to stick together in rouleaux. We do not know exactly how it is that so much hæmoglobin is free in the blood! It has been suggested that the toxin generated by the parasite of blackwater fever is a solvent of the hæmoglobin of even unattacked R.B.C. I look to specialists to enlighten us on this point. May it not be that in blackwater fever where we have the most striking display of blood destruction, we have a special form of sporozoa which has a specially powerful toxin, which dissolves out that on which our life depends, the hæmoglobin. A toxin affecting the hæmoglobin is doubtless generated by all the varieties of *plasmodium malarii*, but individual idiosyncrasy, state of the bodily organism at the moment of attack, the number*

of the attacking host, all tend to determine the form of the particular fever.

What do we know of these toxins?—We know that ptahogenic life by their growth and multiplication generate specific poisonous substances within the system. They must do so. These differ one from the other according to the species of bacteria. By cultivating species of bacteria in nutrient media and then separating these from the media, it is found that there is left a poisonous substance imparted by the presence and growth of the bacteria. These toxins, as far as they have been investigated, are definite chemical bodies.

We do not yet know what the parasite of Blackwater fever really is.—No one can say that he has actually seen the plasmodium in this, as we have done in other forms of malaria. Some affirm that they have seen crescent forms, and some that a black parasite is prevalent, while other observers say that a special bacillus has been found in the urine. None of these observations have been, however, confirmed. Probably the plasmodium is small and only enters the red blood corpuscles and sporulates in the spleen, liver, bone marrow, or brain, and seldom circulates in the peripheral blood. The liver becomes enlarged, and repeated attacks of malaria may render the organ permanently hypertrophied. The intercellular tissue is increased at the expense of the real tissue of the organ. The spleen is similarly affected. It may weigh many pounds. On section the pulp is soft and black, and yellow ochre is abundant. The kidneys show signs of having been frequently inflamed. The brain, too, shows the same symptoms. Sometimes the lumen of the vessels are almost occluded. The endothelium is always swollen and the vessel may be blocked by parasites or melanin. This is exactly what we would expect from the clinical signs—restlessness, tossing of the body, hallucinations, excessive perspiration, abnormal retching, syncope. There seems to me to be such a thing as a quinine hæmoglobinuria, which occurs only in those persons who have been resident in a malarial district for some time, and whose organs have been reduced by malarial fever. Quinine alone does not give it, as recent experiments have demonstrated, but when quinine is given in small doses to a constitution already impaired, it is brought about. This accounts for it not affecting new comers till they have had much fever.

Blackwater fever appears to me to be more prevalent on the highlands of malarial countries than on the lowlands. It is not often seen on the sea coast. It affects young strong men more than the weakly. This may of course be due to exposure. Still, in the former there is always the question of muscular waste. It appears to be most prevalent from January to April—a period of the year when moisture abounds.

During the year (1898) I had seven cases under my care and a word on each may be interesting.

(1) A. B. Been in British Central Africa for four years. Has had blackwater fever twice before. Had only returned from Europe six months. Had been in robust health for months, and had just come off a hunting expedition which lasted four days. He was seized suddenly, in the middle of the night, with what he thought was ordinary fever. He had taken

no quinine for months. Urine very dark but passed freely. It cleared on the third day. He recovered.

(2) C. D., a healthy young planter. Been in British Central Africa six years. Had blackwater fever once before. Had not had ordinary fever for a long time and had been enjoying excellent health. Had been opening up new ground for coffee when he suddenly felt ill. Had not taken quinine for months as he felt in perfect health. He passed urine freely. It cleared gradually on the third day. His previous attack of blackwater fever was also taken when he was engaged in clearing ground and had a similar course.

(3) E. F., a young planter. Had been in British Central Africa for three years. Has had several attacks of ordinary fever during the past few months. The present attack began with severe shivering and vomiting, and the passage of urine like porter, with severe pain in the stomach and testicles. He became as yellow as an orange. On the morning of the fourth day the urine cleared. It continued normal for nine days, but all the time his brain was seriously affected. He died on the 14th day of illness. Probably had been taking quinine.

(4) G. H., a young planter. Been in British Central Africa for six years. Had blackwater fever once before. Had had good health for a long time back. Was working all night pulping coffee. Fell asleep at his work and awoke about 4 a.m. shivering. Severe vomiting, skin not very yellow. Passed urine freely, which cleared on the third day. Made a good recovery.

(5) I. J., Sikh. Been in British Central Africa for fully three years. Had attacks of tertian fever which lasted for three weeks. Two hours after every paroxysm, he passed black urine. Had been much exposed during the rainy season. Previous to attacks had been taking no quinine. He gradually recovered from the tertian fever and the dark urine.

(6) K. L., Sikh. Had been in British Central Africa for three years. Much exposed during the previous two months. Illness began with shivering and vomiting. Passed dark urine in considerable quantity. Yellow. On the third day the urine cleared suddenly. Two weeks after, when marching, had a renewal of the fever, and again the urine became black. This attack had much the same course as before. A month after he had a similar attack with like symptoms. Invalided to India. Had been taking quinine.

(7) M. N., Sikh. Had been exposed to much rain and heavy marching for two months. Had very much shivering. Very yellow. Dark urine. The urine cleared on the third day. Had been taking no quinine.

Besides the above I have had access to notes on two cases of an interesting nature which occurred in my district.

(1) O. P. Had been in British Central Africa for a number of years. Had three previous attacks of blackwater fever. Present attack began with severe shivering and vomiting. Suddenly got yellow. "The urine was almost thick with albumen and blood." It gradually improved, until on the fourth day it became normal and continued so for four days. The amount passed was about 15 ozs. per day. He died on the ninth day.

(2) R. This was a case of suppression. After two days complete stoppage the catheter was passed and a tablespoonful of apparently normal urine was drawn off. He died on the fourth day of illness.

The following should be laid down as axioms:—

(1) *If a man takes blackwater fever before he has been two years in the country he should be invalided home never to return.*

(2) *If he has not taken blackwater fever during the first three years, he has demonstrated that his constitution has a resisting power against this pernicious fever, and that eventually his constitution will adapt itself to the diseases of the African continent.*

(3) *No one should ever treat lightly any form of remittent fever which shows an abnormal temperature of whatever degree, which brings about brain sickness, which is not amenable to quinine, and which shows small parasites of crescent form. At any moment it may develop into a pernicious type of the most serious nature. The bowels, kidneys and skin, excretory organs, chills, should be watched with the greatest care. Let the patient avoid over-exertion, mental worry, fatigue, indigestible food, or any other thing that may upset the balance and tend to depress.*

Treatment.—The literature on the treatment of blackwater fever at the present time presents great differences of opinion amongst writers and physicians practising in the tropics. As I have already said some regard quinine as a specific, whilst very many others regard it as most prejudicial. Great authorities even assert that its administration is at the bottom of all our blackwater fever trouble. I do not believe this; still, I can point, in my own experience, to cases where I think the administration of quinine has done harm, and there are many cases of this kind on record. Whenever a man is threatened with blackwater fever he should, on the appearance of the first symptoms, go to bed at once, keep the skin warm and moist, drink freely of a decoction made from one lemon, sliced, put on the fire in three breakfast cups of warm water, boiled for half-an-hour, strained and sweetened. Hot fomentations should every half hour be applied to the kidneys, liver and spleen. Milk diet should alone be partaken of. Quinine does at least no good, and may do much harm. I can point to a case where blackwater fever seemed to come on when the patient was cinchonised. In some cases quinine acts like glycerine, chlorate of potash, and renders the hæmoglobin unstable. This, superadded to the toxin of the parasite, may bring about the serious blood destruction we have noted in this disease. I would say that in the cases of paroxysmal hæmoglobinuria due to quotidian or tertian fever, with parasites in the blood, the quinine salts should be given, but not otherwise.

The vomiting will certainly be the first thing to call for treatment.—It is a brain sickness. Put mustard on the epigastrium; try 2 drops of liquor arsenicalis every hour; even 4 or 5 minims of the vinum ipecacuanha or $\frac{1}{4}$ gr. of the powder. Cocaine, because of its local anæsthetic effect, may stop vomiting, but often, notwithstanding the brain symptoms, you will be driven to give $\frac{1}{4}$ gr. of morphia hypodermically. When the brain is congested, or there is kidney trouble, it will not act well, and demands the very greatest of care. I see that certain writers on the

bubonic plague find vomiting is controlled by a full dose of calomel followed by sulphate of magnesia.

The second thing demanding attention is the liver.—Continue hot fomentations over this organ every half hour, then purge freely. On the West Coast heroic doses of calomel are often given, say 10 to 20 grs. I usually give 5 grs. calomel and 15 grs. jalap. Some recommend croton oil by the mouth, and turpentine with castor oil by the bowel. I have found the above calomel and jalap, followed in a few hours with an ounce of sulphate of magnesia, to be most effective; it is a grateful drink to the patient, and brings about by osmosis copious watery evacuations. The vomiting controlled, I usually give three tabloids of pigs' bile as prepared by Burroughs, Wellcome & Co., every three hours. I believe the normal bile arrangement has been upset, and pigs' bile being allied to the human is an antiseptic to the poison invading the system. Or a pig may be killed and the bile given *per rectum*. I can point to several cases where it seemed to me to have a marked effect.

Next, in order to help the kidney, I begin at once to give 10 drops of oleum terebinthinæ every three hours. This, in my opinion, acts marvellously. It should be continued till the urine is clear.

After the first serious symptoms are past the danger is relapse. Insist on the patient keeping in bed for at least three weeks. The rest and warmth are equally necessary. Begin the following when the urine clears:—

R. Quininæ bisulph	grains	24
Ac. sulph. dil.	drops	2
Liq. arsenicalis	"	2
Potass chlorat.	"	2
Tinc. ferri. perch.	"	4
Aq. ad.	ounces	12

Signa., a tablespoonful in a wine glass of water three times a day after food.

I see some French writers recommend the following: chloroform 6 drops, sweetened water 250 drops, powdered gum a sufficiency. Of this mixture they give a tablespoonful every ten minutes, until the patient becomes intoxicated. Then the effects are kept up by enemata of choral. I have not tried this.

NOTES FROM SOUTH AFRICA.

By Major M. T. YARR, R.A.M.C.

No. 9 General Hospital, Bloemfontein, May 4, 1900.

THE *personnel* of No. 9 General Hospital—in all 150 officers, N.C.O.'s and men—disembarked at Port Elizabeth on April 4, and immediately entrained with other details in a huge troop train for Bloemfontein. The voyage out from England in the P. & O. transport *Sunda* had been altogether delightful, beautiful weather, an excellent *cuisine*, and the genial courtesy and thoughtfulness of our popular skipper, Captain Andrews, R.N.R., combined to make the trip one of the pleasantest holidays imaginable.

The journey up through Cape Colony to Norval's Poort on the Orange River, through wide plains of

veldt studded with kopjes and peopled mainly by ostriches, was without noteworthy incidence, though interesting to a soldier, owing to the many evidences of recent war—blown-up bridges replaced by trestle bridges, old rifle pits, station sign-boards riddled with bullets, &c.—visible throughout central and northern Cape Colony. At Norval's Poort we were informed that it was quite possible our train would be attacked in the Orange Free State, and all hands were served out with carbines and ammunition. However we reached Bloemfontein early on the morning of April 7 without being molested, forty hours after entraining at Port Elizabeth. Within a few days of our arrival we had pitched our marquees and bell-tents on a pleasant site in front of the Volks Hospital, about half a mile from Bloemfontein.

BLOEMFONTEIN.

Bloemfontein, capital of the Orange Free State, is not, as its name would imply, in the least remarkable for a wealth of flowers, but owes its designation to one Bloem, a Dutch farmer who emigrated from Cape Colony in the early thirties, and settled down with his family and belongings close to the spring which now supplies one part of the town with water. He was soon joined by others, and shortly afterwards the little collection of sod-built houses became known as Bloemfontein (*Anglice* "Bloem's Spring").

The erstwhile hamlet is now a pretty little town of some 6,000 permanent inhabitants. The houses red-brick, tin-roofed little villas with trim gardens radiat-



NO. 9 GENERAL HOSPITAL, BLOEMFONTEIN.

ing from the wide Market Square, in which the Club, Post Office and principal shops are to be found. It is situated in a wide expanse of veldt, sparsely dotted with kopjes, and stands 4,500 feet above the level of the sea. The climate is splendid: dry air, almost continuous sunshine, days warm all the year round, nights cool in summer and cold—occasionally frosty—in winter; for residents in Bloemfontein itself, however, these benefits are to a certain extent nullified by defective sanitation. The dry bracing air, genial sunshine, and high altitude of the country around Bloemfontein, render it almost an ideal place of residence for consumptives, large numbers of whom have immigrated within the last few years. One gentleman,

with whom I am personally acquainted, came here nine years ago suffering from advanced pulmonary tubercle—cavity in right lung, consolidation of the left apex, hæmoptysis, night-sweats and progressive emaciation; he is now a stout, sunburnt man, free from all symptoms, and able to work hard at his occupation, fortunately an open-air one. I have little doubt that when the present turmoil is over many sanatoriums for the open-air treatment will be established in the Orange Free State, and in the neighbouring "Switzerland of Africa"—Basutoland.

At the actual moment of writing an immense body of troops surrounds Bloemfontein, some in tents, the majority in the open; only the Commander-in-Chief, his staff and a few departmental officers live in the town. When it is added that this floating population is at least eight times that of the town, that a large proportion of the troops are mounted, that the town reservoir is cut off by the enemy, that water can only be doled out in small quantities from doubtful wells, and that the "pail system" is the sole means of removing excreta, some conception may be formed of the difficulties the Royal Army Medical Corps and Army Service Corps have to cope with, and the prevalence of enteric fever and dysentery accounted for.

There has been a good deal of desultory fighting close to Bloemfontein ever since our occupation on March 13, with the result that small numbers of wounded are brought in daily for treatment in the military hospitals; but so far, the great majority of cases admitted have been medical ones—enteric fever and dysentery, those curses of armies.

ACCOMMODATION FOR SICK AND WOUNDED.

Three large military hospitals of 550 beds each, No. 8, No. 9 and No. 10 General Hospitals have been established in Bloemfontein; of these Nos. 8 and 9 are marquee hospitals, while No. 10 has taken over various town buildings. Besides these, there are three civil organisations under military control, the Irish, Portland, and Langman's Hospitals, and No. 6, Stationary Field Hospital. In all, we have upwards of 2,000 beds for serious cases. Accommodation for mild and convalescent cases to the amount of 2,000 more is provided in the shape of bell-tent annexes to the general hospitals, and convalescent camps.

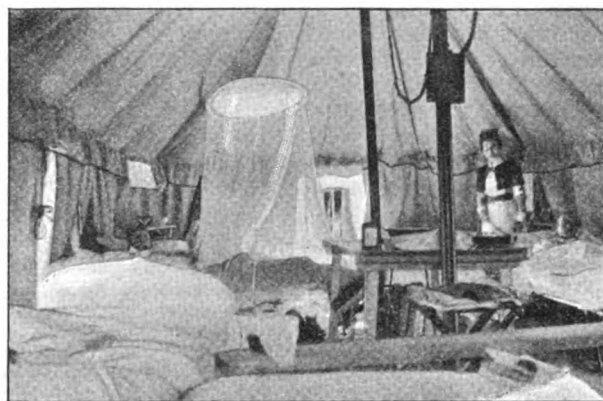
No. 9 General Hospital, the medical division of which I am in charge of, may be taken as a fair representative of a large marquee hospital. The site, a gentle slope running from a long kopje to the railway, is not an ideal one in some respects, but is the best available and has the merit of being close to the railway. On this we have pitched 90 marquees, arranged in parallel "streets" named and numbered, for the accommodation of 550 serious cases, 45 marquees intended for medical, 45 for surgical patients; 41 bell-tents for officers, N.C.O.'s and men of the hospital staff; and 4 marquees with 6 bell-tents for nurses. The hospital staff consists of 18 surgeons—6 of whom are R.A.M.C. officers and 12 civilians—20 nurses, and 134 N.C.O.'s and men drawn from the R.A.M.C., Volunteer M.S. Corps (including 17 students), St. John's Ambulance, and North London Brigade Bearer Company. Behind the hospital proper are 150 bell-tents, in which 600 mild or convalescent

cases are treated pending deportation to convalescent camps. Including this annexe we have thus 1150 beds. Arrangements are made daily for moving a certain number from the bell-tent annexe away to convalescent camps, and for the transference of recovering cases from the marquees to the annexe, thus leaving a number of beds to be filled by the sick convoys constantly arriving day and night. Though my evidence may be considered biased, I cannot



NO. 9 GENERAL HOSPITAL MARQUEES.

refrain from expressing here my hearty concurrence in the general opinion of officers and men of the army of occupation, viz., that the work of the R.A.M.C. officers and the civilian surgeons doing duty with them is beyond all praise; working all day and often half the night, they afford a splendid example of endurance and excellent work done under very trying conditions. Nothing gave me greater pleasure in my



INTERIOR OF MARQUEE, SHOWING FIELD MOSQUITO-NET.

recent visits to Germany and Russia than to note the fact that Continental critics, however severe and caustic in their comments on the causes and conduct of the war, were unanimous in their admiration for the work done by the officers of the R.A.M.C., from the Director-General to the last joined lieutenant.

CASES IN HOSPITAL.

I can only speak of cases in my own hospital, No 9

General; owing to the constant arrival of sick convoys it is difficult for us to leave the precincts of the camp, and anything like an extended inspection of the hospitals in and around Bloemfontein is impossible under present conditions. Within a day or two of opening the hospital we had to take in over 400 serious medical cases, the medical division thus overflowing into the surgical, and at the present moment have only 130 surgical cases, nearly all Mauser bullet wounds. The general impression produced by a visit to the surgical marquees is that the injuries produced are with few exceptions extraordinarily trivial, and that important organs and blood-vessels escape in a way little short of miraculous. The contrast between the aspect and general demeanour of patients who are "only" wounded and those suffering from enteric fever or dysentery, is marvellous; the former cheery, cleanly, helpful to each other, and only too anxious to be at the front again; the latter emaciated, depressed, self-centred, when not delirious. I give details, necessarily of the briefest, of a few of the more interesting surgical cases:—

Case 1.—Captain T., Gordon Highlanders, was wounded in an action about twenty miles away and brought in here last night. The wound was the worst I have yet seen. The bullet entered his left temple, crossed the left orbit reducing the eye to pulp, passed through the ethmoid cells, through the frontal lobe of the brain, thence out through the upper and inner angle of the right orbit, reducing the right eye also to pulp; brain substance was extended through the wound of exit. He was quite conscious and in little pain. Mr. J. W. Smith, a Manchester consulting surgeon who volunteered to come out on our staff as a lieutenant R.A.M.C. (temporary commission), removed the remains of both eyes, inserted a gauze drain in the bullet-track and dressed the whole antiseptically. To-day the patient—an officer distinguished for gallantry in a gallant regiment—is quite cheerful, and is free from headache or other signs of meningitis; should he recover he will owe his life to Mr. Smith's skill and unremitting devotion. Mr. Makins saw the case after Mr. Smith had dealt with it, and entirely agreed with all the steps taken.

Case 2.—Private A., Gordon Highlanders. Bullet entered immediately below middle of spine of left scapula, and came out just above centre of left clavicle. The wound healed without a drop of pus in seven days; there were never any symptoms; and the motions of the arm, shoulder and scapula on chest, are perfect.

Case 3.—Lance-Corporal B., Shropshire Regiment. A nearly spent bullet entered right upper lip, was deflected by the alveolus, came out of cheek at the angle of the jaw (right), finally lodging in the right shoulder, where its exact position cannot be located until we complete the installation of our X-ray apparatus. One bullet thus produced three separate and distinct wound tracts two opening into a septic cavity, the mouth; all the wounds healed kindly within a few days; the motions of the shoulder are perfect and the man is very anxious to rejoin his regiment.

Case 4 and Case 5.—Private C. and Private D. were lying on their left sides in a trench, separated from each other by another man—Private E.—lying in the

same attitude. The enemy managed to get round to the side of the trench and fire down it. A half spent bullet struck Private C. on the ninth rib near the angle, glanced round it, came out five inches farther forward, skipped Private E., the man next to him, and buried itself in the dorsal muscles of Private D. The bullet was extracted from Private D. and both men did well. All three belonged to the Gordon Highlanders.

Several cases of wounds of both thighs by the same bullet were brought in, in no case was the femur or femoral artery injured. The only cases where supuration occurred were two shell wounds, both superficial lacerated wounds, one of the neck, the other of the abdominal wall.

With the exception of forty cases of dysentery, all the patients in the medical marquees are suffering from enteric fever, and nearly all the enteric cases are grave ones. Malaria is, I believe, unknown in this region; and bronchitis, pneumonia, and rheumatism—all diseases one would expect to find amongst men lying on the ground in the open and frequently wet through—are conspicuous by their absence. Cases of sun-traumatism in any form are also very uncommon, notwithstanding the frequently oppressive heat in the day-time and the careless way in which men discard their helmets. Occasionally a man gets severe headache with a little fever from this cause, but as a rule he is well within twenty-four to seventy-two hours.

In all necropsies made on enteric cases so far, most extensive ulceration of Peyer's patches has been found, the ulcers extending down to the serous coat, which at the site of the ulcer is covered by a film of lymph. In every case the tongue is badly fissured and very painful; hæmorrhages from the bowel are very frequent; night delirium also. In fact I have never seen a more malignant type of the disease. A large proportion of the patients have been inoculated on the way out, it is impossible to say how many, in the absence of the men's medical history sheets, which are still at Cape Town.

All the cases admitted under the head of dysentery have been of a very mild type, corresponding to the "catarrhal" form described by Manson: Patient comes in suffering from slight diarrhoea—three or four stools daily—each motion, otherwise nearly normal, having a little blood and glairy mucus mixed with it; no pain, tenesmus, or prostration. With twenty-four or forty-eight hours' rest in bed, milk diet, and a simple astringent, the man gets quite well and returns to his duty. In a few of these cases I have given ipecacuanha in 20 grain doses, with the result that recovery was retarded instead of being expedited; and I am informed by local practitioners that abscess of the liver as a sequela to dysentery originating *de novo* here is unknown. Serious cases of dysentery have been undoubtedly reported from other hospitals here, but I believe in every case they have been recrudescences of dysentery contracted elsewhere. In short, I believe the so-called dysentery occurring in the Orange State bears about the same relation to the dysentery familiar to the practitioner in the tropics as simple diarrhoea does to enteric fever; nevertheless, in the present state of our knowledge of the group of diseases included under the term dysentery, we have

no option but to return such cases of "catarrhal colitis" under the heading "catarrhal dysentery." Manson's words are worth quoting in this connection ("Tropical Diseases," 1898, p. 295):—

"It is well to regard the term "dysentery" as but the name of a symptom or group of symptoms indicating an inflamed condition of the colon—much in the same way as we regard diarrhoea, cough, or fever as symptomatic merely of disease, and not as indicating a single and well-defined disease. Dysentery simply means inflammation of the colon. There may be many kinds of inflammation of the colon."

TRACHOMA AND RACE.

So far, speaking from my own point of view, I regret to say I have seen no cases of eye disease, or eye injuries demanding surgical interference. On the voyage out, however, I was able to elicit some further evidence of the truth of my favourite belief, viz., that trachoma is a disease of "race, not place," and that the pure-blooded negro is practically immune. We touched at St. Vincent, one of the Cape Verde Islands, on the journey out, and by the exercise of a little ingenuity I was able to examine the eyes of some 200 negroes employed at the coaling sheds there. I found among them many cases of corneal nebulae from ulcers, catarrhal conjunctivitis, specific iritis, &c., but not a single case of trachoma, or any of its sequelae, such as entropion, scarring of conjunctiva or old pannus. These negroes all come from the West African coast.

Major Hosie, R.A.M.C., and Captain F. Smith, R.A.M.C., both on the staff of this hospital, have just returned from prolonged tours of service in West Africa, the former in Freetown and the Hinterland up to the Niger source, the latter in Sherbro and the South Mendi Hinterland; both these officers state they have never seen trachoma in West African negroes, though conjunctivitis in various forms is very common. In this connection I would again draw attention to the striking letter from Dr. Renner, of Freetown, published in the *British Medical Journal*, of which I gave an abstract in a recent number of the JOURNAL.

I am indebted to the kindness of Colonel Stevenson, R.A.M.C., P.M.O. Headquarters, for permission to publish these notes. I hope the circumstances under which they have been written will be remembered by readers of the JOURNAL, and accepted as an excuse for their loose and slipshod form.

II.

Bloemfontein, May 19, 1900.

Since I last wrote, the congestion of the Bloemfontein hospitals has been slightly, but appreciably, lessened. Convoys of sick and wounded from the front are as frequent as ever, but the excellent arrangements made by the Principal Medical Officer, Colonel Exham, for removing convalescent and alight cases to convalescent camps, and by relays of trains to Cape Town, are already bearing fruit. A fortnight ago we had 1,750 patients in this hospital and its bell-tent annexe; to-day we have only 1,300, some 500 of which are serious. The work, however, remains hard

and trying, as two of our civil surgeons and four of the nursing sisters are on the sick list. The weather continues delightful, the amount of sunshine and the intense dryness of the air being remarkable.

SURGICAL CASES.

One hundred and twenty-one surgical cases have been admitted within the last week. Of these the vast majority are what we have now learned to call trivial cases, viz., penetrating bullet wounds of the extremities, chest and joints. Some surprise may be expressed at penetrating wounds of the chest and joints being classed under this category, but I can only speak of our own experience; of twelve penetrating chest wounds all recovered without the slightest bad symptom; and the three penetrating wounds of the knee, elbow and wrist joints have healed aseptically, leaving only a daily diminishing slight stiffness. The treatment of all these cases has been absurdly simple and may be summed up in a few words—dry antiseptic dressings to wounds of entrance and exit, rest, no meddling. Fortunately we have so far had no penetrating abdominal wounds to deal with.

Shell-wounds, on the contrary, are much lacerated and slow in healing, but on the whole have done well. Constitutional symptoms in bullet and shell wounds are conspicuous by their absence; the wounded men are cheery, healthy-looking, helpful with each other and all anxious to return to the front—in marked contrast to the emaciated, depressed, leaden-eyed enteric patients.

The following cases present features of interest:—

Case 1.—Hæmatoma of axilla. Private A., Sussex Regiment, admitted on May 6, with a jagged irregular wound about the size of a florin a little external to the inferior angle of the left scapula and a huge hæmatoma distending the corresponding axilla. The tumour was laid open and a large spouting artery—posterior circumflex—tied, a cube-shaped piece of shell some two inches long lying loosely on the musculo-spiral nerve removed; and the whole drained and dressed antiseptically. Patient is doing well, and has had no bad symptoms since operation. This is one instance of many in which important vessels and nerves have had almost miraculous escapes.

Case 2.—Shell-wound of neck. Private B., of the same regiment, was admitted on May 7, with a smallish lacerated wound of the right side of the neck, an inch below the angle of the jaw. A hard body could be felt on the inner side of the sterno-mastoid, about two inches above its insertion; this was cut down on and a small jagged fragment of shell removed from its bed on the sheath of the carotid. Both wounds healed well, and the man is now up and complaining of nothing save slight stiffness of the neck. A similar instance to the last of wonderful escape.

Case 3.—Bullet wound of hip. Pte. C., Gloucester Regiment, admitted May 9, was struck by a Mauser bullet just below the anterior superior spine of the right ileum; the bullet lodged over the anterior crural nerve, causing numbness down the front of the thigh; it was removed and the patient is doing well. Before entering the hip the bullet had passed through a cake of tobacco branded "Lucky Hit," in the man's pocket!

Case 4.—Typhoid gangrene. Gunner D., R.A., was brought in suffering from enteric fever of a fortnight's duration, with gangrene of the right lower extremity extending up to mid-leg, where there was a well-marked line of demarcation. He was in the last stage of emaciation and debility, suffering from constant diarrhoea, and appeared moribund. After twenty-four hours' feeding, stimulants, and rest in bed he had recovered sufficiently to render operative interference possible, and Smith's amputation through the knee joint was successfully performed. For two or three days afterwards he did well, but after that gradually sank from diarrhoea and general exhaustion, dying nine days after amputation. At the necropsy the femoral artery was found plugged as high up as Scarpa's triangle; the intestines were full of typhoid ulcers.

Gangrene is a rare complication nowadays of enteric fever; personally I had never seen a case before, though it is mentioned in the text-books as an occasional epi-phenomenon *vide infra*.

Case 5.—Bullet wound through orbits and brain, destroying both eyes. I described the case of Captain T., of the Gordon Highlanders, in my last communication. It is now only necessary to add that he has recovered without the slightest bad symptom, and is to proceed to Cape Town in two days' time, *en route* for England. It is generally believed that this officer has been recommended for the Victoria Cross for the extraordinary gallantry he displayed in the action in which he was wounded.

Case 6.—Acute cholecystitis. Abdominal section. Death. Private F., Gordon Highlanders, was admitted on May 10 with general peritonitis and stercoraceous vomiting; had felt "bilious" and unwell for a week previously, and was attacked with violent abdominal pain the day before admission. Mr. Smith opened the abdomen, found the gall-bladder distended, the colour of claret, with several ash-coloured oval patches on its surface and adhesions all round to liver colon and small intestine; the adhesions were broken down, and the gall-bladder stitched to the abdominal wound, incised and drained. Patient did well for three days, then gradually sank from peritoneal shock; he was almost moribund on admission, and operative interference afforded the only possible chance of recovery.

All these cases were under the care of Mr. Smith, a Manchester consulting surgeon and lieutenant in the V.M.S.C., who accepted a temporary commission as lieutenant in the R.A.M.C. for the period of the war. Mr. J. W. Smith's services have been invaluable, not only as an unofficial consulting surgeon, but as an able administrative officer in charge of the surgical division: the writer has the medical division, while Major Barrett, R.A.M.C., has the difficult and trying post of managing the bell-tent annexe, a sort of half-way house between the hospital proper and the convalescent camps. The administration of the hospital, no light task, is in the hands of Lieut.-Col. F. E. Barrow, R.A.M.C., and Major A. Hosie, R.A.M.C.

MEDICAL CASES.

An overwhelming majority of the cases admitted to the hospital is composed of enteric fever cases. I

propose to jot down, without regard to sequence or connection, a few of the points that have struck me in dealing with this epidemic of enteric fever.

(1) *Severity of Disease.*—The type of fever is, on the whole, a very grave one and the mortality correspondingly high, but the conditions of warfare—hard work, improper food and exposure during the incubation and initial stages, transport for long distances in ambulances or trains when the disease is declared—must not be forgotten. These conditions are inseparable from military exigencies, no matter how excellent the R.A.M.C. arrangements may be. The severity of the disease as seen in army general hospitals is thus easily accounted for. It is difficult to give an exact estimate of the percentage mortality in this hospital until the campaign is over and the hospital emptied, but so far it cannot have been less than 15 per cent., though I am firmly convinced that the more or less open-air treatment here affords better chances of recovery than treatment under a roof. The mortality in the recent Maidstone epidemic averaged 8 to 9 per cent.

(2) *Recrudescences and Relapses.*—I have only seen one case of true relapse in 365 cases, but "recrudescences" are very common. These recrudescences are so aptly described by Osler (*Practice of Medicine*, 1898) that I quote his description in full:—

"During convalescence, after the temperature has been normal, perhaps for five or six days, the fever may rise suddenly to 102° or 103°, and after persisting for from one to three days, or even longer, fall to normal. With this there is no constitutional disturbance, no furring of the tongue, no distension of the abdomen. These so-called recrudescences are by no means uncommon, and are of especial importance, as they cause great anxiety to the practitioner. They are attributed most frequently to errors in diet, constipation, emotions and excitement of any sort, such as seeing friends." Some of my own cases I attribute to giving patients solid food before the evening temperature has fallen to normal, a proceeding which, though sanctioned by high authority, I now believe to be a mistake.

(3) *Absence of Eruption.*—In the majority of cases the rose spots are conspicuous by their absence.

(4) *Tâches Blenâtres. Peliomata.*—"Tâches blenâtres" on the abdomen and thighs are very common. In every instance body-lice were also present. In this connection the opinion of French observers that these patches are due to the irritating effect of fluids secreted by pediculi is very interesting. Troops campaigning are speedily infested with these disgusting creatures and few, officers, men, or nurses, escape them.

(5) *Fissured Tongue and Sordes.*—I have never seen the tongue so badly affected as in this epidemic. Most of our patients are admitted some days after the onset of the disease, and nearly all have dry, brown tongues, deeply-fissured, and teeth covered with sordes. These symptoms seem to bear no relation to the severity or otherwise of the disease.

(6) *Gastric Symptoms.*—Acute abdominal pain in the region of the stomach is a very common symptom, much remarked on by army surgeons in this campaign. As meteorism is uncommon, and all patients on fluid food, it is somewhat difficult to account for.

(7) *Perforations*.—In three cases patients died of perforation. In two perforation occurred before admission; in the third shortly after; in none did operative interference seem advisable. At the necropsies (*post mortems* are held in all enterics) several ulcers down to, and nearly through the serous coat, were found in the ileum; the perforation was very minute in each case.

(8) *Typhoid Gangrene*.—The single case of gangrene has been alluded to (*vide supra*). The published cases of typhoid gangrene have been collected and commented on by Keen (quoted by Osler); they total 204, viz.: ears 6; nose 10; face, neck and trunk 47; arms 5; genitals 20; legs 126.

(9) *Effect of Inoculation*.—A large percentage of our cases of enteric were successfully inoculated on the way out; I can only rely on patients' statements as the men's documents are in Cape Town. A very grave case, which, however, happily ended in recovery, occurred in one of our own orderlies, who had been inoculated twice by myself. My opinion of the effects of inoculation is only based on my own experience, and at present no one is in a position to compare the percentage of cases of enteric in inoculated and non-inoculated; but I feel bound to say that inoculation, when the operation is only once performed, at all events, is either absolutely useless or we have had to deal with phenomenal cases in this hospital. I must add, too, that hardly sufficient stress seems to have been laid on the collapse, fever and debility following inoculation; the collapse is much more than mere "faintness" and in many cases is alarming; while the fever and debility are such as often to confine the inoculated one to bed for several days.

(10) *Conveyance of Infection*.—Contagion, the *direct* transmission from one person to another, must play a very prominent part in the spread of typhoid in times of war, when we consider that the crowding together of sick in waggons, ambulances, and railway trucks for conveyance to hospital is often unavoidable. On unloading sick and wounded from such vehicles, clothes, kit, and even water-bottles are found soaked or soiled with urine and fæces; while the poor fellows themselves huddle together for the sake of warmth and companionship. Flies, again, are important agents in spreading infection; they constitute a veritable plague here; in the marquees they feast on the sordes of enteric patients and then inoculate themselves in milk or drinking water, notwithstanding every precaution; the British soldier, for some occult reason, has an unconquerable aversion to mosquito nets, and when ill with enteric will take the first opportunity of pulling a net off his bed. To illustrate the extent of this fly-plague I may mention that the larvæ of the *musca vomitoria*, or blue-bottle fly, are exceedingly common in human fæces here; I have often been amused at the serious alarm manifested at this phenomenon by colleagues not familiar with practice in the tropics.

Water and food of course are here as elsewhere the most common modes of conveyance. The modes of conveyance, placed in the following order, afford, I think, a fair idea of the relative importance of the different channels of infection:—(1) water, (2) food, (3) contagion, (4) flies (partly included under 2)

DYSENTERY AND DIARRHŒA.

Since my last communication I have seen no reason to change my views regarding the so-called "dysentery" in the Free State. Acute diarrhœa, with severe colic, is very common, and practically all new-comers are speedily attacked; amongst the troops this complaint is universally known as "the Modders" owing to the severe epidemic which broke out when Lord Methuen's force was encamped on the Modder river.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By GEORGE H. F. NUTTALL, M.D., Ph.D.

Pathological Laboratory, Cambridge.

(Continued from p. 247.)

II.

EXPERIMENTAL AND OTHER EVIDENCE IN RELATION TO THE MOSQUITO MALARIAL THEORY.

ACTING upon a suggestion of Manson's,¹ whose researches on *Filaria sanguinis hominis* had shown the mosquito to be an intermediary host of this parasite, Ross (1895) in India, exposed malarial subjects exhibiting crescentic parasites in their blood, to mosquitoes, and observed the parasites undergoing a metamorphosis within the insect's stomach, similar to that observed on the slide in malarial blood taken directly from the malarial subject. Ross and also Manson, held the opinion (since modified) that the flagellate bodies penetrate into the host, *i.e.*, the mosquito. Whilst examining some mosquito larvæ at Secunderabad (Deccan) Ross observed gregarines² in their stomachs, and concluded that they might represent stages in the development of the malarial parasite. Manson and Laveran naturally considered this conclusion as premature, though Manson (1896) believed that the observations supported his theory of the life history of the malarial parasite. Ross "tracked the germs of this gregarine into the stomach of the mosquito larva, where after an intracellular stage of short duration, and which was not quite satisfactorily made out, it became a large, free,

¹ In reading a historical review of the recent literature on malaria, written by Barbacci (1899), my attention was drawn to a few omissions in the preceding pages. The original publications not being accessible, I must be content to quote the following from Barbacci's paper: "Many authors have raised objections to Manson's theory, or maintained a very sceptical attitude towards it; only Sternberg ('The Malarial Parasite and other Pathogenic Protozoa,' *Ann. Med. and Surg. Bull.*, vol. ix., No. 7, 1897), accepts the theory without reserve. Duggan ('The Parasite of Malarial Fever at Sierra Leone,' *R. Med. and Surg. Soc. of London*, March 28, 1897) on the contrary has no confidence in it, having made the observation in Sierra Leone, where a severe form of malaria prevails, that the mosquitoes only appear during one month in the year, and then are not especially numerous." Anderson (*Ibid.*, February 25, 1896) does not attach any importance to the bites of mosquitoes, claiming that if it were true that these insects carry the disease, Europeans could not survive in the tropics.

² Ross subsequently (1898, ii.) found similar parasites in mosquitoes at Sigúr, also several other protozoal parasites, "any one of which may just possibly be a dimorphic form of the malaria parasite."

actively moving gregarine." At maturity they wandered out of the stomach into the Malpighian tubes, crept to the cæcal end and became encapsuled. Pseudo navicellæ were then formed within the capsule. When the insect has attained the nymph stage, or is fully developed, the capsule ruptures and the pseudo navicellæ escape in great numbers, being given off in the fæces. This was observed in the fully-developed insect whilst in the act of sucking blood. As the mosquito larva devours its own and its neighbours' exuviae it is easily understood how all the mosquitoes in a pool may become infected, and the flying insect might spread the infection from pool to pool. Manson thought this might indicate the mode by which the malarial parasite completes its cycle. At any rate these observations stimulated Ross to continue his studies.¹

Manson (1896, *Lancet*, i., 751) elaborated his theory as follows:—He wrote "As the plasmodium is a passive blood parasite, its escape from the body might be effected on the same principle that the escape of the passive muscle parasites effected. As the latter obtain their opportunity by being swallowed by some flesh-eater—some carnivorous animal—I thought the former might get its chance of development by being swallowed by some blood-eater, some suctorial animal, such as the flea, the bug, the louse, the leech, the sand-fly, or the mosquito." The same idea had already been expressed by Laveran. Manson further drew an analogy between the malarial parasite and the filaria. The filaria is enclosed in a sheath when in the blood, the malarial crescentic parasite is enclosed within the red blood corpuscle, and both parasites when removed from the body leave their enclosing envelope and become motile. This occurs both on the slide and in the stomach of the mosquito. The filaria having cast its sheath, leaves the digestive tract of the mosquito and bores its way into the thoracic muscles, where it completes its metamorphosis. Manson believes that something similar may occur with the malarial parasite, the latter becoming a parasite of the mosquito and entering some cell after the manner of a gregarine or coccidium. The female mosquito having filled herself with infected blood, in due time lays her eggs and dies, her body floating in the water near the egg-raft. The larvæ on escaping from the egg often eat up the dead mother and thus in turn become infected. Manson believed that the malarial parasite may enter the human body through the medium of drinking water, or be inhaled as dust originating from drying up of mosquito-haunted pools, the parasite being in the resting stage. He suggests that the soil may become infected by mosquitoes falling and dying in it. According to Manson then, the mosquito does not produce infection by its bite,

but simply serves as an intermediary host which contaminates the water or soil with resistant forms of the parasite after the latter have undergone a certain metamorphosis in the insect.

Bignami (1896) tried together with Dionisi (1893-1894) to determine if mosquitoes were capable of producing malaria by their bites.¹ For this purpose they liberated mosquitoes, gathered on malarial ground near Rome, in a room in which they placed a healthy man. They made two experiments, both of which proved negative, a fact which they attributed to the dispersion of the mosquitoes in the room and the experiment not being kept up long enough. Bignami states that Calandruccio had observed the malarial parasite to die off both in the stomach of the mosquito and that of the leech. He gives a few of the reasons, given by me in the preceding pages, for his belief that the mosquito inoculates man directly with malaria.

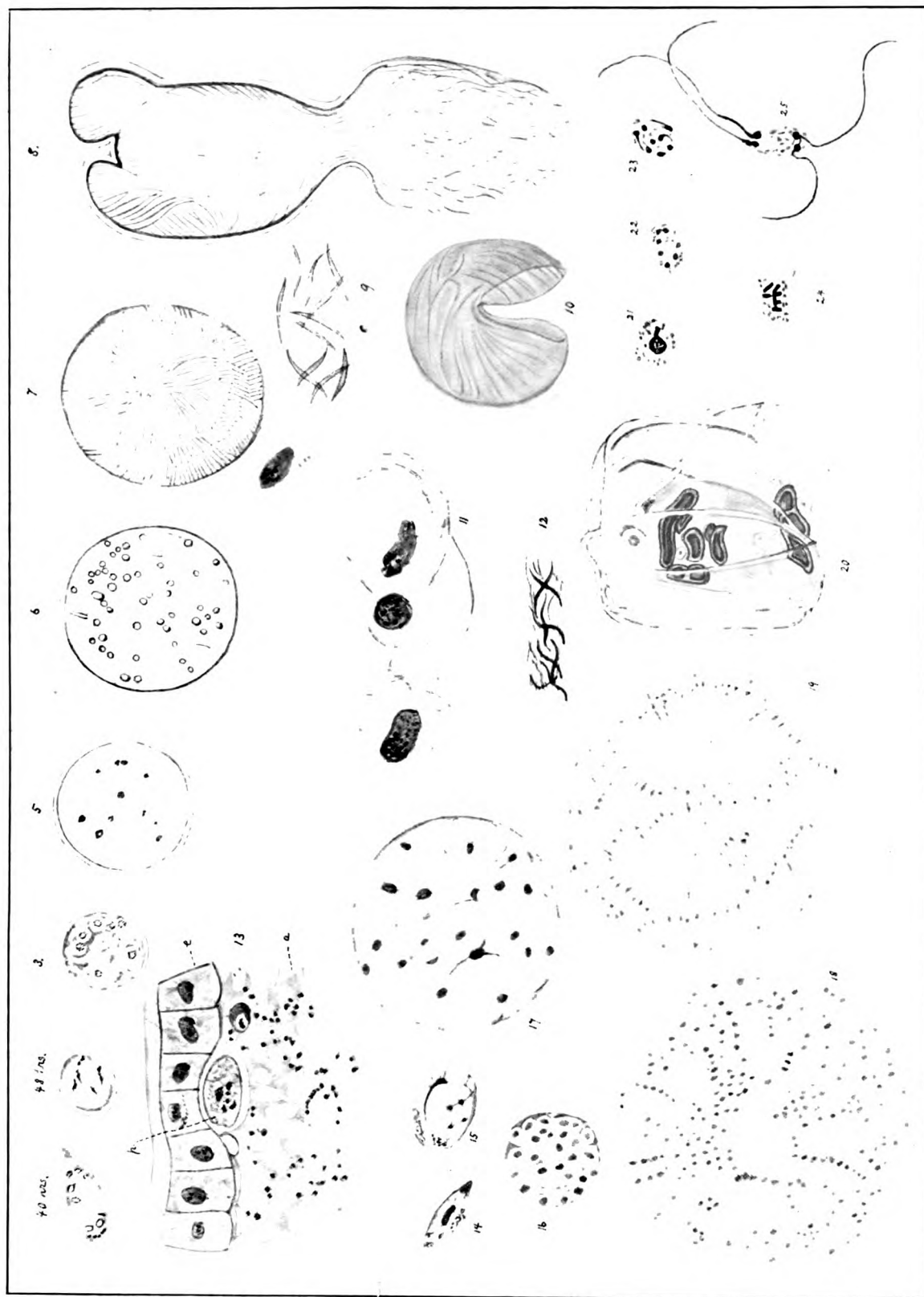
Ross (1897) having continued his observation on mosquitoes, reported that after examining a very large number of these insects he had finally succeeded in finding a few belonging to a particular species² which, after being fed on malarial blood (containing crescents), exhibited certain peculiarities. If he allowed four to five days to elapse after the mosquito had ingested malarial blood, he observed peculiar pigmented cells occurring in the stomach wall of the insect. These cells measured 12 to 16 μ and could be very clearly differentiated from the tissues of the insect. The fact that these cells contained pigment similar to that of the malarial parasite, and that they were not found in control mosquitoes, was certainly highly suggestive. Though, as Ross states, he had previously examined over a thousand mosquitoes with negative result, this positive, or at least suggestive finding, might be explained on the supposition that he had at last found the right species of mosquito to serve as a host for the malarial parasite. Manson had already expressed the opinion that each hæmatozoön probably requires a particular species of mosquito just as in the case of filaria. The cells found by Ross in the stomach wall of the mosquito contained a number of stationary vacuoles, no contractile vacuole, no amoeboid or intracellular movements and apparently no nucleus. All the cells contained pigment granules similar to those of the malarial parasite, the granules, ten to twenty in number, being either bunched together or distributed in lines disposed diametrically or peripherally as in certain hæmatozoa. Some of the granules exhibited slight oscillation. In one mosquito examined after four days, twelve such cells were counted in the stomach wall; in another examined after five days, there were twenty-one cells, but these were more sharply defined and larger than the others. Ross was cautious in drawing any definite conclusions from his work in view of the small number of his observations, the latter, however, gave his subsequent experimental researches a definite direction. Ross's specimens were

¹ Wishing to determine if water might serve as a vehicle for the transmission of malaria to man, Ross allowed mosquitoes to fill themselves with malarial blood, and placed them so that they laid their eggs in the water and died there. The water containing the eggs and grubs of these mosquitoes was given to several natives in quantities of one to two drachms.

Eleven days later one patient developed a malaria, which lasted three days, during which time he exhibited the parasites in his blood. Of course no value can be attached to this single experiment.

² Grassi (1898, ii., p. 237) says these experiments were made with *Culex pipiens*. *Culex hortensis* may also have been used. This would account for the negative result. See further below.

² A description of the species will be found in Ross's article, as also figures of the pigmented cells in the mosquitoes' stomachs.



(Figs. 1-20 after Grassi, Bignami and Bastianelli. Figs. 21-24 after Bastianelli and Bignami, Sept., 1899.)

FIG. 1.—Parasite after 40 hours in *Anopheles* kept at 30° C. It still retains its elliptical form.

FIG. 2.—The same after 48 hours.

FIGS. 3-8.—Progressive development from the third to the eighth day, showing formation of sporozoites.

FIG. 9.—Sporozoites.

FIG. 10.—Empty capsule. (Figs. 1-10 from unstained specimens.)

FIG. 11.—Sporozoites contained in the cells of salivary gland of *Anopheles*. (Stained.)

FIG. 12.—Sporozoites within salivary tubule.

FIG. 13.—Section of stomach-wall of *Anopheles* (schematized). (From stained specimen.)

(p) young parasite, (e) epithelium, (a) adipose tissue of insect.

FIGS. 14-19.—After stained parasites, showing increase in the number of nuclei. (14) After 48 hours, containing one nucleus. (15) After 50 hours. (16) After 3 days. (17-19) After 4, 5 and 7 days, respectively.

FIG. 20.—So-called "brown spores," lying within an empty capsule. (Unstained specimen made from *Anopheles* on tenth day.)

FIGS. 21-25.—Process of flagellation taking place in *Anopheles claviger* and (24) in moist chamber. (From stained specimens, Romanowsky method.) (24) Flagella formed only of protoplasm. (25) The chromatin being contained in the central body. (25) Flagella chiefly made up of chromatin, normal.

sent to Manson in London and examined by him, as well as Sutton and Thin. In a publication which appeared shortly after the above, Ross (1898, i.) expressed the belief that the pigmented cells in the mosquito's stomach wall must be pathological growths, and did not doubt that they were malarial parasites. Ross states that he examined some scores of "dappled winged" mosquitoes unfed or fed with healthy blood; all the examinations were negative, until "at last two of this species were persuaded to feed on a patient with crescents. One of them was killed next day; no pigmented cells could be found. The second was killed forty-eight hours after feeding; numerous pigmented cells were present. They were all small, much smaller than epithelial cells, ovoid, about $7\ \mu$ in the major axis, and each contained about twenty granules of typical pigment which were often arranged circumferentially, just as in the malarial parasite." He also records another observation: "a hundred or more grey or 'barred-back' mosquitoes, unfed or fed on healthy or crescent blood, have been dissected without finding the pigment cells. At last one was observed feeding on a patient whose blood that morning had been seen to contain numerous mild tertian parasites." The mosquito killed on the third day contained many pigmented cells measuring 8 to $25\ \mu$. Manson had supposed that the flagellated malarial parasite developed in the stomach of the mosquito might, after the manner of the filaria, gain access to the tissues of the insect. After Ross's publication Manson (1898, i.) wrote: "As the flagellum carries no pigment, if the pigmented cells belong to the mosquito, or extracorporeal, phase of the malaria parasite, how account for the pigment these cells contain?" He considers that MacCallum's recent discovery (*Journ. of Experimental Medicine*, January 7, 1898) apparently supplies the explanation. MacCallum found in the case of halteridium infection in birds, as also in the æstivo-autumnal malarial parasite of man, that the function of the flagellum is to impregnate the pigmented spheres. In the case of halteridium the impregnated spheres, after a short period of rest, became converted into locomotive vermicules which carried the characteristic pigment granules at one end, whilst with the other hyaline extremity they are capable of penetrating and destroying leucocytes, and apparently by the slightest contact to injure the envelope of the red blood cells, so that these exude their contents into the surrounding serum. MacCallum was not able to observe the formation of vermicules in the malarial parasite of man. Manson, however, believes that such vermicules are formed by the malarial parasite in the body of the mosquito, and that the vermicules wander into the stomach wall of the insect, giving rise to the pigmented cells observed by Ross.

(To be continued.)

YELLOW FEVER IN MEXICO AND CENTRAL AMERICA.

—A severe outbreak of yellow fever is reported from Vera Cruz and along the southern coast line of Mexico and the Central American States. The heat is stated to be abnormally severe in these districts at present.

THE KAFFIR AND HIS TOBACCO.—There are (says a correspondent of *Tobacco*), many varieties of tobacco cultivated in South Africa besides the well-known Transvaal leaf, and the principal of these are the Pondo, Xesibe, Basuto, and Fingo. These tobaccos are grown in the different native territories from which they take their name. The Pondo tobacco, which is supposed to be the king of Kaffir tobacco, is grown on the sea coast. It is made up in the shape of a cone, the leaves being damped and pressed together. The cone is then packed into a covering, made of reeds gathered from the river banks, and the whole is then offered for sale at the trading stores. This Pondo tobacco always commands a good price, the average being about 2s. per lb. It is a pure tobacco and is largely smoked by up-country white men, but is very strong. The Xesibe tobacco, which comes next in popular estimation, is made up in long, thin rolls, which are also encased in reeds. This tobacco is not so full flavoured as the Pondo, and is accordingly less prized, for the up-country resident, as a rule, likes his weed to be fairly powerful. The average price paid for this tobacco is 1s. 3d. per lb. The other two tobaccos are much cheaper, and are only smoked by the lowest class of natives.

FOR AMŒBIC DYSENTERY.—Dr. Aderhold says that the most successful way of destroying the amœbæ seems to be by giving medicated enemata, of which a large variety is permissible; e.g., quinine sulphate, 1-5,000 to 1-1,000; bichloride of mercury, 1-10,000 to 1-5,000; hydrogen peroxide, 1-20 to 1-5; silver nitrate, 1-3,000 to 1-1,000; zinc chloride, 1-2,000 to 1-1,000; methyl blue, 1-1,000 to 1-100. The position of the patient while taking the enemata may vary, but if he lies on his back, leaning to the right side, with his hips elevated a foot or more, he will find it a suitable position, which he can maintain for some time without being exhausted. The enemata should be given once or twice a day and may be interchanged to suit the individual case. Dr. Jay found it a decided advantage to alternate the quinine with peroxide of hydrogen on different days, while at the Augustana Hospital it was interchanged with methyl blue.—*New York Medical Journal*, March 31.

SULPHUR AS A PREVENTIVE OF MOSQUITO BITES.

—One of our readers informs us that, having seen a statement in some English medical journal to the effect that sulphur, taken internally, would protect a person against flea bites, it occurred to him to try it as a preventive of mosquito bites. Accordingly, he began taking effervescent tablets of tartaric acid and sulphur, four daily. He provided himself with several lively mosquitoes and, having put them into a wide-mouthed bottle, inverted the bottle and pressed its mouth upon his bare arm. The mosquitoes settled on his skin, but showed no inclination to bite him. If this gentleman's experience should be borne out by further trials, it might be well for persons who are particularly sensitive to mosquito bites to take a course of sulphur during the mosquito season, especially in view of the growing opinion that the mosquito is the common vehicle of the *Plasmodium malarie*.—*New York Medical Journal*, May 19.

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THE

Journal of Tropical Medicine

JUNE, 1900.

TYPHOID FEVER IN THE SOUTH AFRICAN ARMY.

IN an earlier article it was pointed out that ill-health would likely affect the troops when the stress of the Boer campaign had had time to make itself felt. In making this forecast, though anticipating much loss from disease, we were hardly prepared to hear of the great mortality from typhoid fever which is now affecting the army in South Africa. The wars to which this generation have been accustomed were so comparatively short in their duration that there was little chance of the development, to any great extent, of the diseases which, in former times, were the ordinary accompaniments of large armies in the field. Still even in such short wars as the Franco-Prussian, smallpox ravaged the French

army and was not sparing of the German, and led to that great pandemic of smallpox among the civil population of neighbouring countries which distinguished the seventies; nor was the Russo-Turkish war free of typhus fever in either armies, though its after-effects appear not to have been very great. These might have served, in a certain degree, as warnings to the possibility of the diseases of campaigns not being extinct. Possibly more attention would have been paid to this fact were it not that few imagined that the Boer war would be anything but short, and if it were prolonged that the great advance in sanitation in the English army since the Crimean war would be unable to cope with the dangers that might arise. The war, now happily within measurable distance of its termination, has lasted eight months, and in that time experience has proved contrary to many expectations. First of all the war has not been a small one; over 200,000 men have been required to invade the country, three towns have been subjected to the horrors attending prolonged sieges, stubborn battles have been fought and forced marches have been made which have caused the death of thousands of horses. These things have been done in a country whose inhabitants, according to report, are not given to cleanly habits. The conditions are such as not to be favourable to the purity of the soil, water or air, and we are now beginning to note the effects in the long lists of deaths from typhoid and dysentery which are published in the official statements and progress reports.

The deaths in Ladysmith from typhoid fever, dysentery, and other diseases, were extraordinarily numerous considering the duration of the siege. They were, however, taken as a repetition of the history of all besieged towns, where sooner or later disease becomes rampant and destroys more soldiers than either the guns or assaults of the enemy. Anxiety and insufficient food play their part in the causation of disease, rendering those subjected to their influence an easy prey to the surrounding physical and insanitary conditions, injurious to health under ordinary circumstances, but peculiarly so under the abnormal conditions at the time of a siege. The large number of

deaths among the besieged at Ladysmith, and the 800 cases of typhoid fever found in the hospital on the day of General Buller's entry into the town, bear witness to the sufferings which the beleaguered garrison underwent during those weary months while waiting for relief. Could any of this mortality and sickness have been prevented, is a question which only those present and most conversant with the conditions could possibly answer. We believe everything was done that could possibly be thought of, and which the exigencies of the siege permitted. Besieged towns have little choice, and the only measure which proves permanently successful is early relief. But while we are inclined to think that the deaths from typhoid fever at Ladysmith were unavoidable, it is impossible to view the deaths from typhoid fever that are occurring elsewhere in the same light. Mr. Fripp, we see, states in a letter sent from South Africa to the *British Medical Journal*, that at the time of writing there were nearly 2,000 cases of typhoid fever in Bloemfontein. We have lately counted the number of deaths in the South African army from typhoid fever in one of the official lists of deaths from disease recently issued, and we find no fewer than 100 deaths from typhoid fever occurred between May 30 and June 6, and of these no fewer than 23 occurred on June 4. It is obvious from these figures alone that the army is suffering from an epidemic of typhoid fever, and since the deaths are not reported from one station but from many stations, that the infection is wide-spread. The difficulties connected with its prevention are great. Doubtless the disease is liable to be carried by infected soldiers from one place to another, and in that manner healthy localities are apt to become affected, while the primitive sanitary arrangements of the towns must favour to an immense extent its extension. The position is one therefore which requires the most active measures to check the disease and preserve the lives of our soldiers from a preventable disease.

PLAGUE.

PLAGUE is so widely spread at the present moment that its future progress is well nigh impossible to predict. Since its appearance in Hong Kong in 1894, the disease has been met with in every continent, although, fortunately, with the exception of Asia, the outbreaks have been very mild and localised. In Europe, Oporto is the only city where the disease has prevailed, and it is now many months since any case of plague has been reported from Portugal. In North America a few cases of plague have been met with amongst Chinese immigrants on the Pacific shore, but up to the present there is no indication that the disease is obtaining a hold on the community.

In South America plague first declared itself at Asuncion, an inland town, the capital of Paraguay, during 1899, but no epidemic ensued, and the Paraguayan authorities have just pronounced their republic free from the disease. At Santos, in Brazil, and on the La Plata river several towns have had a few cases, and the disease still lingers there, but in a limited and a mild form.

In Africa plague is said to be endemic in the Hinterland of Uganda. In South Africa imported cases of plague have been met with at several ports, but no outbreak has occurred, nor are there at the present moment any known cases of plague in South Africa. In Egypt, Alexandria was attacked by plague, but although the disease lasted several months the outbreak proved mild in the extreme. For well nigh a year Egypt has been free of plague, but lately as many as fifty cases have been notified in Port Said, and three or four cases at Suakim.

The Australian continent, until within the last few months, had never been visited by plague, but the city of Sydney is suffering from an inroad of the disease which threatens to be serious. As yet no great mortality has ensued, but at Adelaide, Melbourne, Brisbane and Rockhampton, several cases of plague have occurred.

Asia is the great field of plague. India, well nigh from north to south and from east to west, is more or less seriously infected. Bombay, ever since

the disease first appeared in 1896, has never been rid of plague and the fourth epidemic is only now on the wane. Calcutta, where plague appeared at first in a strangely sporadic form, has within the last six months suffered from an epidemic of the disease, and at the present moment the mortality from plague, although the severity is lessened, amounts to thirty or forty per day. In other parts of India, the north-west provinces, the city and district around Patna in Bengal, and at Kura-chi, plague is prevalent at the present moment.

From Teheran plague is reported, and many districts of Arabia, more especially on the Red Sea, are affected. Eastwards, in Southern and Northern China, Hong Kong, Japan, and in Siberia in the district around Lake Baikal, plague is evidently endemic.

The Islands of the Pacific are being gradually attacked. The Philippines, Honolulu and New Caledonia are all declared infected by plague, and it seems but a question of time before the inhabitants of many other islands fall victims to the disease.

In Asia the Malays and the Burmese seem to be able to protect themselves, for as yet neither of these races have contracted plague.

Lastly, in Mauritius, although the disease is no longer epidemic there, sporadic cases occur, with a persistency which betokens endemicity.

Reply to Article for Discussion.

DOES CANCER OCCUR IN NATIVE RACES (OF INDIA)?

THE above subject for discussion will come as a surprise to all who have had even a small experience in India, where all kinds of malignant growths are common enough. The museum of the Medical College of Calcutta contains a large number and variety of such growths, the great majority of which have been obtained from natives of this country.

In order to test the subject further I have been through the *post-mortem* records of the last twenty-two months at the Medical College,

Calcutta, during which time 450 *post mortems* have been performed on natives alone, among which sixteen had died of malignant disease, twelve having been cancerous and four sarcomatous; so that 1 in 28 deaths, or 3.55 per cent., were due to malignant growths. Further, I find that out of the 450 cases no less than one-third were due to such diseases as malaria, dysentery, cholera, and plague, which would not complicate the returns of deaths in England to any appreciable degree, while the expectation of life and the proportion of persons living at the cancerous age is certainly much lower than at home, so that it is difficult to compare the cancer rate of the native of India with the English rate. At any rate the figures just given are sufficient to show that malignant diseases not only occur in natives of India, but they may be said to be common among them.

LEONARD ROGERS, M.D., M.R.C.P.,
Offic. Professor of Pathology,
Medical College, Calcutta.

THE BERKEFELD FILTERS.

At a time when our attention is being forcibly and all too sadly drawn to the prevalence of enteric and other intestinal ailments in South Africa, there needs no apology for bringing to prominent notice some of the various appliances for purifying water used for culinary and drinking purposes. The Berkefeld Filter holds a pre-eminent place in the public estimation, and in our opinion deservedly so. The Government has entered into a contract with the Berkefeld Filter Co., for sufficient filters to supply every man now on active service in South Africa with filtered water. The filter in question has been specially constructed for the War Office under the auspices of the Army Medical Department, and is officially described as the Berkefeld Field Service Filter.

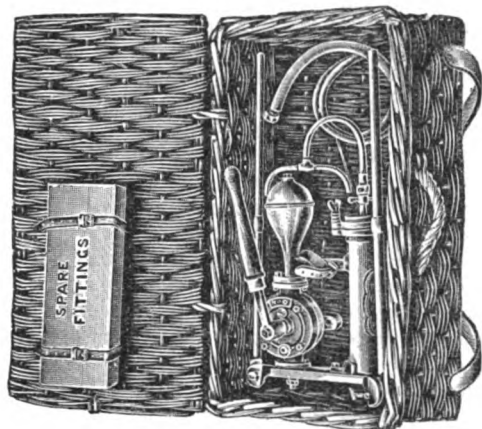
THE BERKEFELD FIELD SERVICE FILTER.

Description.—The "Berkefeld Field Service Filter" consists of the filter proper and a semi-rotary brass pump mounted together on a tripod stand with folding steel legs: for transport the filter is packed in a wicker basket which can be strapped to the pack saddle.

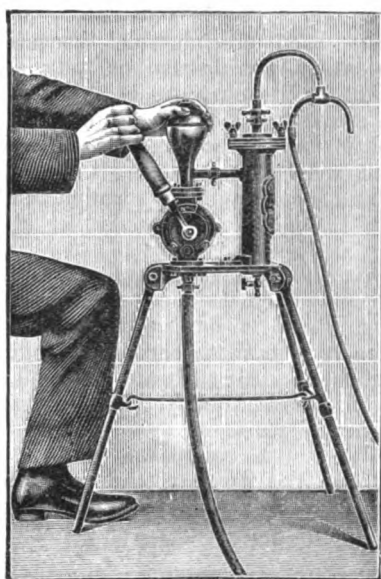
The Berkefeld Field Service Filter has been constructed in such a way to give filtered water for a unit of 100 men.

Water pumped through the Berkefeld Field Service Filter is guaranteed to be absolutely free from any disease-bearing germs.

Filtering Medium.—The filtering medium in the Berkefeld Filters is in the form of hollow cylinders made of kieselguhr, a substance which, on account of its enormous porosity, consisting as it does of the silicious skeletons of diatoms, is an ideal filtering medium.



How to Use the Filter.—Work the handle of the pump at the rate of about twenty to thirty strokes per minute; in this way the water to be filtered, which is drawn up through the suction hose from a bucket or other vessel, is forced through the filter and the filtered water is discharged through the two outlet tubes direct into the water bottles.

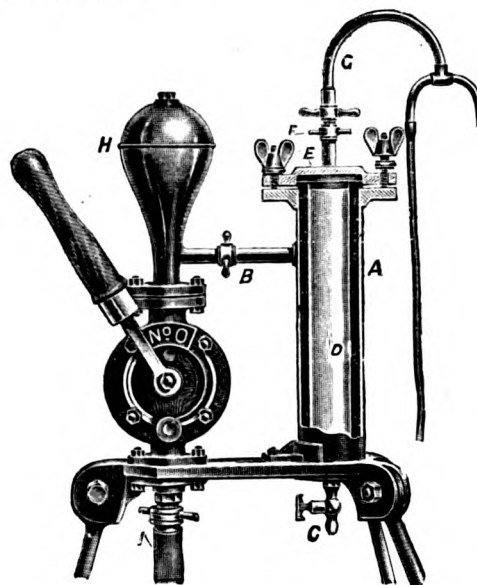


When and how to Clean the Filter.—According to the quality of the water to be filtered the filter will require more or less frequent cleaning, and as all the impurities collect on the outside of the filtering cylinder, producing a slimy coating, the filter itself will indicate when it requires cleaning by a diminished output and by the greater force which is necessary to work the pump. To clean the filter proceed as follows:—

Take the filtering cylinder out of the filter case

and give it a good brushing all round with a piece of the loofah supplied for this purpose, or with a moderately hard brush, and afterwards rinse with clear water. Care must be taken that no soap, oil, or grease of any sort comes into contact with the filtering cylinder.

From time to time the cylinders must be put into cold or tepid water and boiled for about thirty to forty-five minutes.



THE TRAVELLER'S PUMP FILTER.

This filter is a compact and handy combination of a suction and force pump with the filter proper. The most essential part is the filter cylinder A, a hollow cylinder fitted with a bent outlet tube K, and secured in the filter chamber C by means of the lugged screw cap B. This cylinder being breakable should be handled with care, and on no account must the screw nut at the bottom of the cylinder be interfered with.

The suction end E of the filter, which is detachable, consists of the foot with the nipple-shaped inlet, above which a small piece of wire gauze is placed to catch the coarser impurities, and which screws into the upper part containing the suction ball valve. The whole is attached to the lower part of the filter chamber by means of the milled screw nut D. (The wire gauze must be cleaned occasionally by means of a tooth brush.)

Just above the lower inlet ball valve there is another ball valve which admits the water drawn into the pump by the upward movement of the piston G into the filter chamber C upon the downward movement of the piston.

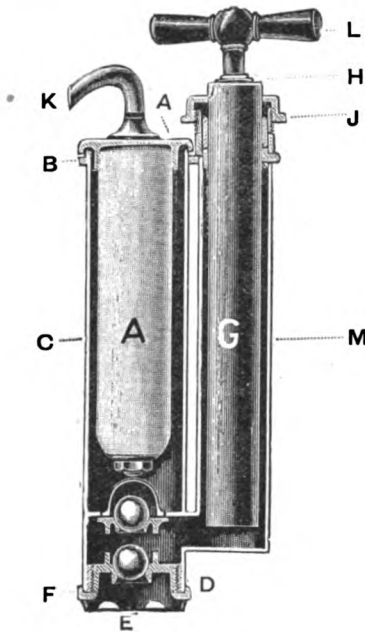
The stuffing box H, which requires re-packing from time to time with asbestos cord or cotton, is secured by means of the screw cap J to the upper part of the pump.

For lubricating the piston vaseline or tallow should be used but no oil.

The joints at B and F are formed by an india-rubber or dermatine washer placed into proper grooves provided for this purpose.

Before using the filter take out the filtering cylinder, blow through the suction end to remove any dust from the packing that may have lodged in the valves, and clean the filter chamber C with a damp rag, or by rinsing with clean water.

To Use the Filter.—First see that the filtering cylinder is firmly fixed by means of the lugged screw cap, and that the bent tube K is turned in the direction shown in the accompanying illustration, viz., away from the pump handle. Grasp the filter round the middle, rest the suction piece E firmly on the bottom of the receptacle containing the water, draw up the piston to its full length, and force it back again to the end of the stroke. By this operation a column of water is drawn into the case M and is driven through the upper ball valve into the filter chamber C, where, finding no other outlet, it is forced through the pores of the filtering cylinder, and leaves by the bent tube in a highly purified state.



A new or perfectly dry filtering cylinder should be allowed to soak in water for about half an hour before being placed in the filter case, and the first portion of filtered water should be thrown away as this has a somewhat earthy taste.

A piece of india-rubber tubing may be slipped on to the nipple-shaped suction piece to draw the water direct from a river or a pool if no vessel is at hand.

When leaving off filtering it is advisable to loosen the lugged cap B a little (spanners for unscrewing are supplied with each filter). This will prevent it sticking and will also save the india-rubber washers from being quickly worn out.

To Clean the Filter.—As, owing to the density of the filtering medium, all the impurities from the water collect on the outside of the filtering cylinder, all that is required to clean the filter is to carefully remove the cylinder from its case and to brush it with a piece of loofah, supplied for this purpose with

the filter, or a medium hard brush, and afterwards rinse it with clean water. Care must be taken that no oily or greasy substance comes into contact with the cylinder.

It depends entirely on the quality of the water one has to deal with how often the filter has to be cleaned; as a rule this should be done every day, especially in hot climates. However the filter will tell its own tale, as in the degree as the filtering cylinder becomes coated with impurities the filtration will become slower, and considerable force will be required to get any water through. About once a week the filtering cylinder should be placed in cold or tepid water, and boiled for one hour in order to perfectly sterilise it. It is recommended to have one or two spare cylinders in reserve.

The cleaning operations should not be entrusted to native servants.

Filters supplied to travellers are provided with waterproof canvas cases with strong leather shoulder straps, holding pump; or strong tin boxes, holding pump and two spare cylinders, with arrangement for padlock (box can be used as sterilising vessel).

Reprints.

ACUTE DYSENTERY.

By GEORGE A. HUNTLEY, M.D.

DEFINITION.

THERE has been considerable discussion as to whether dysentery is a disease or only a symptom of disease, but for the purposes of this paper we may define it as a disease of the large intestine, attended by inflammation of the solitary glands and follicles, and characterised by frequent stools containing blood and mucus, and accompanied by tormina and tenesmus.

ETIOLOGY.

Among the principal etiological factors, predisposing and exciting, may be mentioned:—Bad or insufficient food, impure water, a diet wanting in vegetables, alcoholism, mental anxiety, excessive fatigue, tropical climates, and chill. All ages are prone to dysentery, and there is no race immunity. Malaria has probably some place in the cause of dysentery, but this is a disputed point. The complaint prevails more extensively during the autumn and summer months. The actual etiology of dysentery is unknown, by some it is attributed to the presence of amœba, by others to bacteria.

CLINICAL FORMS.

There are three clinical forms, namely, acute catarrhal dysentery, acute tropical dysentery, and acute diphtheritic dysentery.

MORBID ANATOMY.

In studying the morbid anatomy it will help us to remember that the mucous membrane of the large intestine has the poorest blood supply of any mucous membrane. In acute catarrhal dysentery of a mild type the lesions are confined to the lower portion of

the large intestine; in severe cases, however, the whole of the large bowel may be involved, and even a portion of the ileum. The most prominent feature in this variety is the enlargement of the solitary follicles, which in severe cases may slough and leave a large number of small round ulcers, which may coalesce and cause larger ulcers of an irregular shape; these are, however, usually confined to the mucosa. In the tropical form the condition is much more serious. Here the floor of the ulcer may be on the floor of the mucosa, the muscularis or the serosa. The edges are infiltrated or undermined. "Nor does the surface ulcer give anything like an adequate idea of the amount of mischief done by the disease to the intestinal tissue, for the ulcers often undermine the mucosa, coalesce and form sinous tracts bridged over by apparently normal mucous membrane. The cæcum, hepatic and sigmoid flexures, and the rectum are particularly liable to be involved, but in very severe cases the whole of the large intestine may be thickened and reddened with ulcers, leaving only a few solitary islands of intact mucous membrane." During the healing process there is formation of fibrous tissue at the base and sides of the ulcers which later contracts and may result in partial stricture of the bowel. "In the diphtheritic form of the milder grades the follicles of the colon are capped with a thin yellow exudate. In severe forms the colon is enormously enlarged, the walls are thickened, stiff and infiltrated, and the mucosa from the ileocæcal valve to the rectum represented by a tough, yellowish material. It is an extreme necrosis of the mucosa." (Osler.)

SYMPTOMS.

The disease is usually preceded by a preliminary diarrhoea, lasting from one to three days; more rarely, however, the onset is sudden. The more severe forms commence with a chill or distinct rigor usually. The principal symptoms are frequent passages, presence of blood and mucus, and tenesmus. With these symptoms will be associated tormina, tenderness on pressure, and the stools will have the peculiar dysenteric odour. If the disease has its seat at the upper part of the large intestine, altered biliary secretions will be mixed with the blood and mucus. If the dysenteric process is confined to the lower portion of the bowel, the blood will be separated from the mass and will occur in streaks. There will not usually be more than eight to twelve discharges in the twenty-four hours, though there may be as many as thirty. Fever is variable; some cases may be afebrile. A careful examination of the pulse is of much greater importance than the use of the thermometer—a rapid weak pulse being an exceedingly grave symptom, while a high temperature need not necessarily cause alarm. There will be tenderness on pressure in most cases after the first few days, and usually progressive loss of strength and flesh. Nausea and vomiting are occasionally observed. In nearly all severe cases the microscope will reveal the amœba actively moving. As the disease advances in the severe type the stools change in character, marked by the absence of faecal matter and containing sloughy threads of exudative material looking like "washed raw meat," mixed with blood and pus. Pulse is frequent, small, and feeble,

and the thickened intestine may be felt through the abdominal wall. The tongue becomes dry with brown or black centre and red edges. Restlessness and mild delirium. The urine is dark and scanty; there may be strangury. There is a cold sweat, and the patient generally passes into a drowsy torpid condition. In speedy cases consciousness may be retained to the end, but in protracted cases there is low delirium, succeeded by collapse, coma, and death.

DURATION.

Duration is anywhere from a few days to several weeks. Malignant dysentery may terminate fatally in two or three days. Death may occur from exhaustion, from hæmorrhage, from perforation, peritonitis, or from secondary pyæmic abscess. In all cases, however, the immediate cause of death is asthenia.

COMPLICATIONS AND SEQUELÆ.

Among the complications and sequelæ may be mentioned liver abscess. This is a formidable complication, and in India occurs once in every four or five cases. Another—perforation—usually fatal. More rarely, intestinal stricture. And as an interesting sequel to dysentery may be mentioned paralysis; many cases having been reported occurring in the form of a paraplegia, due to a neuritis.

PROGNOSIS.

The prognosis in simple, acute, or catarrhal dysentery may be considered good, but in the asthenic malignant, or scorbutic type exceedingly bad.

TREATMENT.

In the discussion of treatment we come to the most interesting and the most important part of our subject. To correctly diagnose dysentery will not be difficult in the great majority of cases, and whether the solitary follicles are principally affected, or some other interesting portion of the anatomy, will not immediately concern us in consideration of the case. A patient with an acute and dangerous disease has entrusted his life, humanly speaking, in the hands of the attending physician. The all-important question is, What shall be done? Addressing medical men it is not necessary to say that the days of treating acute dysentery with opium, lead, and other astringents, have happily passed, and we have adopted more rational and successful methods. The researches of bacteriology, though they have not definitely shown to us whether the disease is caused by a coccus, a bacterium, or an amœba, have proved beyond all reasonable doubt that a germ of some kind or other bears the principal part in the etiology of this complaint. Opium has imprisoned that germ in the intestinal canal, has masked the true picture of the disease, and sudden death has often occurred when friends regarded the case as doing well. The most rational treatment will be topical. The patient should be required to keep the recumbent posture and exhorted to give way as little as possible to the desire to go to stool, and co-existing conditions as malaria or scorbutus recognised if present. The topical treatment will be two-fold, and consists in purgatives and enemata. One-drachm doses of the salines may be given frequently, or corrosive sublimate gr. 1 in 100

every two hours. The writer has found the combination of salol and calomel very successful, especially in children, though in the case of his own child the turning point in a severe case of amœbic dysentery proved to be in the administration of hydrarg. perchlor. strength 1 in 10,000, one drachm every hour. Purgatives should be combined with the use of salol, 10 grains every four hours for adults and proportionately small doses for children. It will be necessary to watch the urine for symptoms of carbolic acid poisoning. When blood and mucus cease to appear in the stools and the discharges have become feculent in character, it will be best to leave off the purgatives and administer intestinal antiseptics alone. The salicylate of bismuth and salol will probably be best at this stage. If the character of the stools do not change after a fair trial of these remedies, ipecac. should be tried, either in large doses of 30 to 40 grains, or in smaller doses of $\frac{1}{4}$ -grain every half hour. In the administration of ipecac. the strictest quiet must be observed, and it should be given while fasting; the first thing in the morning being a particularly favourable time. Preceding the treatment with small doses of cocaine or nepenthe will diminish the nausea, or a mustard plaster may be applied to the stomach for the same purpose. The new preparation, "Ipecac. sine emetin," has not proved so efficient as the ordinary ipecac. powder of the B.P. or U.S.P., and I do not think that ipecac. has proved itself to be the "Radix anti-dysenterica" in China as it seems to have been in India, though it undoubtedly is useful in many cases.

ENEMATA.

The bowel should be washed out after every evacuation with warm water and quinine 1 in 5,000, 1 in 2,500, or 1 in 1,000. Three to six pints of fluid may be used in the adult. A serious obstacle in acute cases will be found in the irritability of the rectum and the tenesmus which follows any attempt to irrigate the colon. This may usually be overcome by the injection of a small quantity of a 4 per cent. cocaine solution or a cocaine suppository. The long rectal tube should be used and the stream applied slowly. The patient should be placed in the dorsal position with a pillow under the hips for the purpose of gravitation. When the acute symptoms have passed, the injections are well borne and are soothing. Instead of quinine, boric acid or permanganate of potassium may be used.

For the abdominal pain use turpentine stupes, hot fomentations, or hot-water bottle. The writer has usually employed locally ol. sinapis 1 part to 4 of olive oil with good results. Occasionally, but very rarely, a hypodermic of morphine may be called for.

Diet is all important, and should be confined during the acute stage to milk, barley water, egg albumin, beef juice, and broths in small quantities and nearly cold. Special care should be given to diet during convalescence, as the appetite often returns before the digestive organs have recovered their tone, and a lack of care at this time may result in a serious relapse. Corn flour and arrowroot will be permissible at this stage. Koumyss will be palatable to some, while most will appreciate "cod fish gravy," made

from the dried codfish procurable at most of the stores, which is boiled in milk and strained and a small quantity of flour added. During the period of convalescence iron, bitter tonics, and mineral acids will be indicated.

All reports of the topical and rational treatment of dysentery show a considerable diminution in the mortality, and cases which formerly lasted for six or eight weeks are now often cured in as many days.—*American Baptist Missionary Union, Han-Yang.*

British Medical Association.

ON THE PREPARATION AND USE OF CALMETTE'S ANTIVENENE.¹

By O. W. ANDREWS, M.B., D.P.H.,
Staff-Surgeon, R.N.

VARIETIES OF POISONOUS SNAKES.

POISONOUS snakes are chiefly met with in warm countries. All tropical countries except the Pacific Islands contain poisonous snakes, and most of the temperate regions, with the exception of New Zealand, do also. Venomous snakes are divided into two principal classes:—

(1) *Colubrine*.—For example, cobras (Asia), moccasins, and coral snakes (North and South America respectively), black snake, tiger snake, and deaf adder (Australia).

(2) *Viperine*.—For example, small viper (England), rattlesnake (North America), chain viper (Daboya), russelli (of India), and puff adder (of South Africa). Colubrine snakes have comparatively long maxillary bones, which are mobile, but to a less extent than the corresponding bones of viperine snakes, and ankylosed to the fore part of the maxillary bones are grooved fangs; posterior to these teeth in some varieties there are smooth hooks without furrows. Viperine snakes have broad heads, very short and very mobile maxillary bones carrying fangs which are perforated by a complete canal, which alone would serve to distinguish a viperine from a colubrine snake. The poison apparatus consists of a gland homologous with the parotid salivary gland of other vertebrates; it is situated on each side of the head behind the eye and in front of the tympanic bone, which is united by ligaments with the mastoid bones and mandibles; the gland is so placed beneath the masseters and internal pterygoid muscles that when the fangs pierce the skin the contraction of these muscles ejects the venom through a duct opening at the base of the poison fang.

COLLECTION OF THE VENOM.

The method employed at Lille to collect venom is as follows:—

The snakes are kept in large iron-wire glass-lined cages in a house warmed artificially, so that the

¹ Much of the matter included in this paper is contained in an unpublished report furnished by me to the Director-General of the Medical Department of the Navy in a course of instruction given at the Pasteur Institute, Lille, by the Director of that Institute, Dr. A. Calmette, to delegates from the Navy, Army, and Indian Medical Services.

temperature never falls below 27° C. (= 80.6 F.). When a snake is required, it is grasped with the right hand below the head with catch forceps about three feet long (resembling tongue forceps). It is next caught between the index finger and thumb of the left hand. The jaws are then forced apart by the thumb and forefinger holding the snake, care being taken that the mouth of the snake is turned from the operator and his assistant, lest some of the venom be injected into the eye, as it is readily absorbed by the conjunctiva, and even a very small amount suffices to set up severe ophthalmia. The fangs are next cleared by pushing back the sheath of mucous membrane which surrounds them, and the assistant holds a watch-glass beneath the poison fang; whilst the person holding the snake encourages the ejection of the venom by pressure on the glands with his (free) right hand. When the secretion ceases to flow the assistant removes the watch-glass, and inserts a glass funnel down the mouth of the snake, breaks a raw egg into it, and forces this through the funnel by means of a glass rod. The snakes appear to resent this artificial feeding, but it suffices to keep them alive and well. The collection of venom and feeding being completed, the snake is returned, and the process repeated in about a fortnight's time. In returning the snake the catch forceps are always employed in the same manner as when taking it out of its cage.

M. Calmette, as a rule, mixes the venom from all the snakes in his collection, which, when we were there, included amongst others the *naja naja* of Egypt (Cleopatra's asp); cerastes or horned viper, also of Egypt, and the *Pseudechis porphyriacus* (black snake) of Australia, *Hoplocephalus curtis* (tiger snake) from the same country, *Bothrops lanceolatus* (fer de lance) of Martinique, and *Crotalus durissus* (rattlesnake) of North America.

COMPOSITION OF SNAKE VENOM.

Venom, which is always collected from a fasting snake, is a clear limpid fluid resembling white of egg in consistence but of a light yellow colour; it is acid in reaction, and has a specific gravity of 1050. The average yield is from 2 to 3 c.cm. according to the size of the snake. A good-sized cobra yields from 30 to 45 mg. of dried venom. The venom is dried at a temperature of 16° to 20° C. in a desiccator over calcium chloride, and when dry has the appearance of gum arabic. If thoroughly dried and kept in well-corked bottles it preserves its toxic properties indefinitely. The composition of venom has been investigated by Dr. C. J. Martin, who finds that it consists of coagulable and non-coagulable proteids, and that the coagulable proteids may be regarded as albumoses rather than as globulins, the albumoses being hetero-albumose, proto-albumose, and deuto-albumose.

Snake venom is not a simple poison but is composed of at least two distinct poisons; one of these, the one which has the power of producing hæmorrhagic extravasation and intra-vascular clotting, is destroyed by heating a solution to 85° C. But there is another, or others, not destroyed by a temperature close to 100° C. If a solution of venom be heated to a temperature of 102° C. for twenty minutes all

toxicity is completely destroyed. Dr. C. J. Martin states that if the fluid be filtered after precipitation of the albumen by heat varying between 70° and 95° C., and the filtrate dialysed in a current of sterile distilled water, the venom thus freed from albumin and deprived of its salts still contains a substance in the dialyse which, when dried rapidly *in vacuo*, consists of a brown amorphous powder forty times more toxic than normal desiccated venom, and which, moreover, kills in a manner which shows all the classical symptoms of snake poisoning. The analogy existing between the toxic albumoses produced by bacilli and those produced by the epithelial cell of the venom glands of snakes has been shown by Dr. Sydney Martin. The experiments of MM. Calmette and Deléarde show that the blood of animals immunised against abrine is antitoxic for the venom of serpents as well as against ricine and diphtherial toxins, so that the production of immunity against snake-bites is quite as feasible as against these vegetable toxins. M. Calmette has shown that the only reliable chemical reagents are solutions of hypochlorite of lime 1 in 60, freshly prepared, containing 80 per cent. of available chlorine, or else 1 per cent. solution of gold chloride. Both of these reagents injected into the tissues immediately around the wound are capable of destroying any venom with which they come in contact. *In vitro* potassium permanganate can exert the same power, but in contact with the tissues of the body this power is speedily lost, therefore permanganate is not of any practical value. It throws down a dark precipitate of albumen when mixed with a solution of venom. Permanganate mixed with venom solution which has been previously heated to 80° C. and freed from albumen by filtration still throws down a precipitate, which, when dried, consists of a brownish powder. Ammonia, alcohol, chloroform and mercury perchloride all precipitate the albuminous bodies from venom, but these precipitates are capable of being redissolved in water or in excess of the reagent, and are as toxic as pure venom.

SYMPTOMS OF SNAKE POISONING.

These vary with the species of snake concerned, the local effects being slight in some cases and grave in others.

Cobra Bite.—The symptoms are swelling of the part with intense pain and redness. The swelling extends towards the body; there is intense congestion, and the patient becomes drowsy or faint, suffers from vertigo, great weakness of the legs, profuse salivation, and loss of speech, but still appears to be conscious. There is dimness of sight, an aspect of extreme anxiety, nausea, vomiting, feeble pulse, and laboured breathing. The patient lies on his back, the respiratory efforts grow feebler, then the breathing ceases. Convulsions due to asphyxia follow, and lastly the heart ceases to beat, respiration stopping before the heart. Until respiration ceases the pupils are somewhat contracted, but respond to light.

Rattlesnake Bite.—There is considerable pain and swelling at the seat of the wound, and the local injuries have to be reckoned with, even after the general symptoms have ceased to cause anxiety.

Indian Viperine Snakes.—Bloody discharges from

the rectum and other orifices of the body are very noticeable symptoms and ecchymoses occur in various organs; albuminuria is observed during recovery, which is never the case with cobra poisoning. Another distinct feature is that the pupils are always dilated and insensible to light.

TREATMENT.

Ammonia.—Professor Halford's method consisted in injecting 10 or 20 drops of the strongest ammonia, mixed with 20 drops of water, into the jugular or median vein, a method which Sir T. Lauder Brunton long ago pointed out was powerless to protect the victims of the very venomous snakes. Frank Buckland was a great believer in ammonia as a remedy for snake bites, but Calmette discourages the use of either alcohol or ammonia, which he says only do harm.

Strychnine.—Dr. Müller, in Australia, injected strychnine hypodermically in all cases of snake bites until the physiological action of the drug was manifest. Dr. L. Ralston Huxtable, of Sydney, N.S.W., at the Intercolonial Medical Congress of Australia, Sydney, in September, 1892, gave statistics of 426 cases of snake bites as follows:—

	Number.	Deaths.	Mortality.
Cases treated by strychnine	113	15	13.2
Cases treated otherwise	313	18	1.4

Calmette's Serum.—M. Calmette, in February, 1894, gave an account of the treatment of snake bites by means of the serum of an animal immunised against cobra venom, and also against that of a viper, and against that of the Australian tiger snake. Experiments were first conducted by producing immunity in rabbits by the injection of gradually increasing doses of venom, commencing with a dose well under the lethal; and, finally, injecting doses many times the lethal. The method of producing the serum is as follows: A solution is first made from the dried venom of three distinct species of snakes, viperine and colubrine (*Crotalus durissus*, *Bothrops lanceolatus*, and *Naja tripudians*); 3 grammes of dried venom, the estimated cost of which is £6 sterling, are dissolved in 300 c.cm. of distilled water, the solution is effected by frequent agitation in a flask during a period of twenty-four hours, the solution during the intervals being kept in a dark cupboard. After this it is placed in three flasks with flattened sides and heated for thirty minutes in a water bath, the temperature of which is kept at 72° C. by means of a Roux's regulator. Heating to 72° C. coagulates the albumin, which falls down as a flocculent white precipitate, which is removed by filtration through filter papers. In all experiments hereinafter mentioned, solutions of mixed venom prepared in this way were employed, namely, 1 in 100 for injecting into all animals except guinea-pigs, for which 1 in 1,000 was used.

The horses employed for the production of anti-venomous serum are strong, young, healthy cart horses kept in stables hygienically perfect. All horses employed must be proved to be free from glanders by giving a negative reaction with the mallein test. The neck is washed with carbolic lotion. Half a milligramme of venom (that is $\frac{1}{2}$ c.cm. of 1 in

100 solution) is injected into the subcutaneous tissue just in front of the shoulder. An abscess follows in a few days; it is opened and dressed with antiseptic lotions. After fifteen days, if the abscess has healed, 1 mg. (that is, 1 c.cm. of solution), is injected, and at the end of another fortnight 2 mg. are injected. The dose is increased from time to time, so that after one year the horse can resist 0.5 gram (that is, 50 c.cm. of solution) when injected subcutaneously, a dose which would suffice to kill twenty-five horses not previously immunised. After sixteen months of this treatment the horse should be sufficiently immunised to yield a valuable serum, and after two or three years it is immunised to the extent of being able to resist a dose of 1 gram (100 c.cm. of solution). When this state is reached the serum is said to have the strength of 200,000 units according to Roux's notation, that is to say, that if a rabbit be injected with a dose of serum equal to $\frac{1}{200,000}$ of its weight, it will resist a dose of venom capable of killing the same weight of rabbit in twelve hours; 1 c.cm. of anti-venomous serum injected intravenously should protect a rabbit against a dose of venom calculated to kill a similar but unprotected animal in twenty minutes. Immunity produced by repeated injections of venom is comparatively evanescent, so that horses after they have been brought up to the maximum degree of resistance must be injected every two or three months with 1 gram of dried venom (100 c.cm. of solution). When employing large doses of solution of venom for horses, a wide-mouthed bottle is used, having a rubber cork perforated for two tubes which are arranged as in a wash bottle, the short tube is connected with a Higginson's syringe, the longer one which reaches to the bottom of the bottle is connected with a rubber tube terminating in a metal cannula which is introduced into the subcutaneous tissue of the neck in the situation already described, the solution being injected by bringing the syringe into play.

The method of bleeding the horses is similar to that employed in obtaining anti-diphtherial and other serums. The serum, after being allowed to separate in a dark cool room for twenty-four hours is syphoned off into 10 c.cm. bottles fitted with rubber corks and caps; sterilisation is effected by heating to 60° C. in a water bath for one hour on three successive days. This temperature is sufficient to destroy any micro-organisms which might possibly have gained access to the serum during collection, but is insufficient to interfere with its protective power.

It is important to note that a temperature of 60° C. does not in any way impair its protective value, as this is a higher temperature (140° F.) than would ever be met with in the shade anywhere. The antitoxic value and also the superiority of intravenous over other modes of injection are clearly shown in the experiments which were performed by Major D. Semple, R.A.M.C., Major R. W. S. Lyons, I.M.S., and myself.

Reviews.

THE EARLY TREATMENT OF APPENDICITIS. By Dr. Donald Hood. Post-Graduate Lecture. Messrs. Bale, Sons and Danielsson, Limited, 83-89, Great Titchfield Street, London. 38 pages. Price 2s. 6d.

This lecture will serve as a useful guide to the practitioner and help to remove much of the unjustifiable prejudice against the use of opium in appendicitis. We are fortunately emerging from the stage of treatment in which surgical interference was always called for if the patient had 500 dollars in his pocket, and bringing rational therapeutics into a sphere of illness from which it has been excluded for many years.

Dr. Hood points out that we meet with appendicitis as a fulminating peritonitis due to perforation of the appendix, or as a less acute form of inflammatory action localised to the ilio-cæcal fossa. Perforation may in some cases give rise to no premonitory symptoms; there may be no sickness, no pain, no tumour to be felt in the right iliac fossa, and the tumour containing pus may be met with on the left side of the abdomen. The less acute form of appendicitis may be mistaken for typhoid, especially when no tumour is to be felt, and a commencing attack may simulate most of the acute abdominal ailments—such as acute intestinal obstruction, renal colic, gall-stones, gastric ulcer, diarrhoea, and other gastric symptoms accompanying influenza. The four cardinal symptoms of appendicitis are—pain, pyrexia, local tumour and immobility of parietes, with functional disturbances which cause diarrhoea, or constipation and frequently vomiting. It is impossible to say at the outset whether the appendicitis is of the suppurative or the non-suppurative form; in fact, many consider supuration a mere complication and not a condition arising as the natural sequence of the disease. Dr. Hood insists upon full doses of opium being given from the first in the treatment of appendicitis. Opium lessens pain, checks peristalsis, procures sleep and enables the patient to do with less food. At the same time the patient should be kept in bed, fed on milk and broth, and have hot fomentations to the abdomen. Aperients are neither necessary nor expedient.

Every practitioner of medicine should have this book at hand for reference and for guidance in cases of appendicitis.

RECOLLECTIONS OF MY LIFE. By Surgeon-General Sir Joseph Fayrer, Bart., K.C.S.I., LL.D. William Blackmore and Sons, Edinburgh and London, 1900. Pp. 508.

To review this book in the ordinary way seems altogether unsatisfactory, and to me impossible. I took up the book one evening at 8 p.m., and by the following evening at 8 p.m. I had read every word of the book from beginning to end. Engagements and professional work seemed but to intrude during the twenty-four hours, and the few hours of sleep were grudged, as they prevented my progress with the story. And a wonderful story it is, for not only is it

the sketch of the life of a great and distinguished man, but it is told as it were from within, confiding all, confessing all, lightly touched yet deeply affecting, a unique account of an exceptional career.

Of course the great event associated with Sir Joseph Fayrer's life is the fact that he was residency surgeon at Lucknow during the siege. There is nothing comparable to this siege in history, and there is no account of the siege to be compared with that which has just issued from Sir Joseph Fayrer's pen. I have visited Lucknow; I have seen the residency, Fayrer's house, the Moti Mahal, the Alum Bagh, &c., and on the very ground gone over the details of the siege, but have never read any account that brings the facts of the siege so keenly home to one as that given us by Sir Joseph Fayrer. The soldier sees but a limited portion of the battle, and little, it may be, of the sick and suffering; the surgeon's sympathies are, on the other hand, widely distributed. Deaths from disease and injury by missiles are but the ordinary accompaniment of battle, but when threatened starvation adds its horrors, when cholera, smallpox and typhoid hover around, and when surrender means death not only to the men but to every woman and child, and these one's own, the ordeal passed through by medical men in Sir Joseph Fayrer's position is beyond conception.

The early part of the book is a distinct contribution to history. The Sicilian rebellion, the fighting in Rome during the Revolution of 1848, are told with an insight and circumstance within the knowledge of few other Englishmen. This book will be highly valued by not only the medical profession, but also by the historian. From the medical military aspects of these Recollections there is much to be learned. An observer so astute as Sir Joseph Fayrer, an original investigator so thorough in his methods and a writer of such eminence, was calculated to produce a book of intrinsic interest, and he has done so.

I would wish to add a further important recollection of intense interest to all who wish this Journal well. Sir Joseph Fayrer wrote the first article for the JOURNAL OF TROPICAL MEDICINE, and although it may appear but a small matter to him, I believe it will prove a fact of more than mere platonic historical interest. I repeat again, I have spent the best part of twenty-four hours in reading to a finish every word of this interesting work, and I am convinced that those who take up the book will be constrained to follow my example.

J. C.

News and Notes.

HORSE SICKNESS IN SOUTH AFRICA AND MOSQUITOES.—Chief Surgeon Kuhn claims to have completely solved the question of horse sickness in German South-west Africa. He declares that he has proved it to be a "kind of malaria" communicated by mosquitoes. Inoculation experiments, Dr. Kuhn says, have been wholly successful. That horse sickness may be caused by mosquitoes is quite possible, but that it is a "kind of malaria" is a statement of so loose a nature as to scarcely merit serious notice. True malaria is a rare disease in South Africa, but

what "kind of malaria" it may be it is impossible to say.

RESISTANCE OF MICRO-ORGANISMS TO INTENSE COLD.—In the *Proceedings of the Royal Society*, vol. lxi., No. 427, an account of Professor Dewar's experiment shows that even the intense cold brought about by exposure for twenty-four hours to the temperature of liquid air does not destroy the vitality, or even impair the subsequent activity of the following micro-organisms on solid and liquid media: *Bacillus typhosus*, *Bacillus coli communis*, *Bacillus diphtheriæ*, *Spirillum cholerae Asiaticæ*, *Bacillus proteus vulgaris*, *Bacillus acidilactis*, *Bacillus anthracis* (sporing culture), *Staphylococcus pyogenes aureus*, *Bacillus phosphorescens*, and *Photobacterium vaticum*. The fact that the vitality of these micro-organisms was unimpaired was tested after the medium containing them was carefully thawed. After this experiment it is easy to understand how cholera continues to be virulent during even a Russian winter. In the dwellings of the Steppes all stools and house refuse are thrown out on the snow surrounding the dwelling houses in winter. The source of the water supply of the household, with all the wells, &c., frozen, is the snow from the neighbourhood of the house, and from the fact that the cold and snow do not impair the virulence of the cholera bacilli it is easily understood how cholera continues to spread in a Russian household during the severest winters.

QUERY FOR ASSAM READERS—PIPSAS.—What are the pipsas mentioned at page 59 of "Recollections of my Life," by Sir Joseph Fayrer. He refers to them as having been met with during a journey in Assam, and describes them as a sort of mosquito, which leave little blood-stained marks wherever they bite. Perhaps some of our readers in Assam can inform us?

THE CAUSE OF SCURVY.—The sum and substance of the communication made to the Royal Society by F. G. Jackson and Dr. Vaughan Harley, the result of an experimental enquiry into the cause of scurvy, may be summarised as follows:—That the absence of fresh vegetables (or lime juice) is not alone sufficient to cause scurvy, nor is their presence sufficient to prevent or cure the disease; but that the condition of the food in general, and more especially the state of preservation of the meat supplied, must be regarded as the primary agent in the production of scurvy.

PLAGUE IN FISH.—A new propagator of plague has, it is said, been discovered at Tokyo. After a local flood a quantity of dead fish were thrown up on the shore. It was found that the fish caused illness, and on further investigation it was shown that the fish were infected with the plague bacillus. Rats fed on the fish quickly sickened and died with all the signs and symptoms of plague. Of marine animals crabs alone have hitherto been proved to be attacked by the plague bacillus, but the fact that fish may become widely infected is disquieting indeed. [We cannot

give any authority for this alarming statement. The paragraph occurred in a Japanese newspaper.—EDITOR.]

Letters, Communications, &c., have been received from:—

C.—Dr. H. Clare (Jamaica).
H.—Dr. W. Walter Hoare (British N. Borneo).
M.—Dr. Macvicar (Blantyre).
N.—Dr. F. Neal (Mahaica).
P.—Dr. Paterson (Grenada).
R.—Dr. Duncan J. Reid (Shanghai).
S.—Mr. Edw. Sutton (China).
Y.—Surg.-Major M. T. Yarr (Bloemfontein).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

- 1.—All communications will be acknowledged in the *JOURNAL* under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.
- 2.—Manuscripts sent in cannot be returned.
- 3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.
- 4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.
- 5.—Authors desiring reprints of their communications to the *JOURNAL OF TROPICAL MEDICINE* should communicate with the Editors.
- 6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

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In order to meet the constant enquiry for addresses of respectable firms catering for the various requirements so difficult to obtain abroad, we give a list of names and addresses which we trust will be found useful to our numerous correspondents and subscribers.

Original Communications.

CAMP FEVERS AS SEEN AT MASSOWHA BY ITALIAN OBSERVERS.

By Dr. FILIPPO RHO.

*Privat-docent of Medical Pathology (Univ. of Rome);
Surgeon-Major of R. Italian Navy.*

THE question of tropical fevers is often connected with that of camp fevers in these climates, and both are most intricate. That the argument is still a matter of confusion and doubt, not only practically but also theoretically, is shown by the discussion that took place on the subject of tropical diseases at the meeting of the British Medical Association, held at Edinburgh in 1898, with reference to a communication made by Colonel Crombie.

Since then several remarkable papers have been published by our American colleagues, to whom the campaign at Cuba had offered an ample field of observation; and since, as Morgagni said, *nulla est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias tum aliorum tum proprias collectas habere et inter se comparare*, may I be permitted here to draw, as succinctly as possible, a parallel between the fevers studied by us Italians during our occupation of Massowha and those studied by the Americans at the time of their war with Spain for the conquest of Cuba.

The observations made by the Italians embrace the period from 1885 to 1894; and although at that time the diagnostic resources for studying the typhoid infection were not what they are now, yet it may be asserted that the discussions which took place and the conclusions that were arrived at, were identical

to those the Americans recently came to; therefore they corroborate each other.

I believe Massowha to be one of the most interesting tropical regions with regard to the study of fevers in hot countries, as it is situated under the highest isothermal line, is thickly populated by a barbarous indigenous race, and is free from malaria—in fact, in the discerning and appreciating of other morbid factors, it is a considerable advantage not to have to take into consideration this tenacious infection that so often complicates the tropical disorders. Perhaps this was the reason why, even without the help of the latest discoveries in diagnosis, it was possible for us to come to pretty safe conclusions from the very first years of our occupation.

The attention of our medical men out there was at once drawn to the extraordinary number of cases of fevers which broke out in extensive epidemics, especially in the less dry and less torrid season. These fevers declared themselves suddenly, mostly without shiverings, and the symptoms thereof were: pains in the forehead, loins, and eyes, an exceedingly foul tongue, but never dry nor cracked; loss of appetite and constipation in most cases; the skin either dry or perspiring, according to the temperature, which hovered between 38° and 40° C. The fevers of some duration went down daily about one degree or little more, either in the morning or in the afternoon, but they never showed an intermission. In most cases the patients were affected, sometimes at the beginning, sometimes in the course of the malady, by a measly rash about the neck, chest, and back, and on the extensor surface of the limbs. Rarely any diarrhœa, and then always moderate, with a slight splenic enlarging in cases where the fever was protracted beyond a week; no other localised disorder.

A slow convalescence, accompanied by an exhaustion quite disproportionate to the brevity of the malady.

This, then, is the general and almost uniform nosological description of these fevers. In duration they, however, presented the most varied features. From very short ephemeral fevers lasting twenty-four hours, to such as protracted themselves to three weeks and more; all the intervening gradations were noticeable. Most of them were of a brief duration, and the patients got over them in about seven days. Very seldom did they last two or three weeks or more, assuming a decided look of typhoid fever, but sometimes, in fresh epidemical outbreaks of similar endemial fevers, typical cases of the typhoid fever were more frequent and more easily recognisable by the clinical and anatomopathological characters.

Some of these fevers which, contrariwise, were marked by constipation and few or no abdominal symptoms, took a long and indefinite run, entirely resembling the Mediterranean or Malta fever, and were generally cured by the patient returning to the native country.

Much has been said on the nature of these endemial-epidemical fevers of Massowha, and especially about those most numerous cases of a week's duration or little more. At first it seemed that finding in a tropical land fevers of a continuous remittent type, should strengthen the opinion of many that remitting fevers of a malarial nature were in question. At first I thought so myself.

But Pasquale's careful examinations of the blood proved that the hæmatozoa of Laveran was not to be found in those febricitant patients; besides, the geological and topographic conditions of Massowha—which is situated on a small madreporic island—are contrary to the development of the malaria, as there are no marshes, either great or small, in which the *anopheles* or malaria mosquito prospers. Others, like Petella, give it as their opinion that these are climatic fevers *a calvie*, Pasquale calls them rheumatic fevers *a frigore*, others again an infection *sui generis*. Some even maintained that most of these fevers, even the mildest, were due to Eberth's infection; but the practitioners opposed to them the well-known laws of Wunderlich which, in truth, were not taken much into account by these same fevers. Some speculative bacteriologists of those times spoke of the typhoid bacillus and the bacillum coli being identical, and of the latter being transformed into the former by the effect of heat. Some hygienists went so far as to gainsay the existence of typhoid fever in such cases, not knowing how to explain the appearance at Massowha of such an infection, judged, after the current scholastic theories, to be generated only by water, whereas our patients had only drunk distilled water.

It was then I combated all these oppositions with arguments which, to repeat them again, would be like beating the air; and I concluded that all the fevers seen at Massowha, except a few cases of ephemeral fevers, should be ranged among the diseases of a typhoid order, and thus classified according to their growth: gastric fever or mild typhoid, real typical enteric or typhoid fever, and mild protracted typhoid (*febbicola tiphoide* of the Italians). I have now nothing more to say regarding this classification,

except that some of these slight fevers called protracted typhoid fevers are most likely really Mediterranean or Malta fever of the kind now recognised by Bruce and Hughes.

The question, however, chiefly concerns the vast number of mild forms which last a week or thereabouts, are accompanied by cephalalgia and constipation, and nearly always end by lysis.

In hot climates typhoid fever generally runs a less dangerous course than with us, the abdominal symptoms are less serious, and the ulcerations often absent; it is therefore natural we should there meet with an enormous number of cases of abortive typhoids, or what Griesinger denominates *typhus lævissimus*, or *tiphoidette* according to Brouardel's and Lorain's appellation, which stand to the typhoid fever as the chicken-pox stands to the small-pox (Jaccoud), a fact we already observe on a smaller scale in our own climates. In fact Kelsch and Riener have demonstrated, after a careful study of a number of documents and the clinical registers of military hospitals, that the frequent febrile gastric disorders which are to be looked upon as abortive typhoids, follow the exact course of epidemic fevers.

According to my knowledge there already existed, with regard to Massowha, a case bacteriologically well ascertained by Pasquale; indeed, he had succeeded in isolating from the blood the bacillus of Eberth in one of these short mild fevers, and only refrained from somewhat generalising out of respect for scholastic traditions and the laws of Wunderlich, and ended by giving the origin of the endemial fevers of Massowha a rheumatic character.

The progress of bacterioscopic diagnosis and diagnosis by means of serum test afterwards confirmed the existence of similar forms of sickness in other places. In Italy, Lucatello and Silvestrini had already isolated, the one from the spleen, the other from the circulating blood, the bacillus of Eberth in so-called gastric fevers lasting only four or five days, and which are very frequent with us, especially in summer time. In analogous cases Silvestrini, Fontana and others, often obtained a positive Vidal's reaction; others again, like Lazarus and Brigg, &c., were able to isolate the typhoid bacillus from faecal matter by the Elsner process.

Hence I think that there can be no further doubt on this head, and that many of the fevers, as well in tropical as temperate climes, which are being attributed to various causes, and are called climatic, must be ascribed to typhoid infection.

NOTES FROM SOUTH AFRICA.

By Major M. T. YARR, R.A.M.C.

No. 9 General Hospital,
Bloemfontein,
June, 15, 1900.

Now that Bloemfontein is no longer the "front," surgical cases in the hospitals are becoming few, and an overwhelming majority of the beds are occupied by enteric fever cases. "No. 9" is no exception to the general rule, there being only some sixty surgical cases in a total of over a thousand patients. Enteric

fever is still very rife, though the type of the disease is perhaps no longer quite so severe. This diminution in severity is probably due to the fact that more patients are brought to us now by rail and fewer by ambulances and buck wagons.

ENTERIC COMPLICATIONS.

A few brief notes of complications, not mentioned in my previous communications, or only briefly alluded to, may be of interest.

Typhoid gangrene.—I have now seen three cases of this rare complication, one affecting the leg (described in my last), one of the penis, and one of the cheek. In the case of gangrene of the penis, the fever had from the first been of a severe type, with high temperature and delirium; towards the end of the third week the penis swelled slightly; within forty-eight hours a regular oval greyish-black patch, quite dry, appeared on the dorsum; the patch was about the area of a florin and clearly demarcated. The patient died two days later, just as the slough was beginning to separate; the fever was high, up to a couple of hours of death, which was apparently due to typhoid toxæmia, as there were no other complications. The patient with gangrene of the cheek was admitted a month ago and was probably in the second week of fever on admission. He was a man of weakly physique, and the disease ran the usual course of a moderately severe case, defervescing gradually till the temperature fell to sub-normal ten days ago; by that time he was a mere skeleton, and a bed-sore had formed over the sacrum notwithstanding every precaution. For three days the temperature remained below 96.4° morning and evening, and the extremities were with difficulty kept warm. Seven days ago his right cheek swelled slightly—a diffuse swelling involving the entire cheek pretty equally; this he himself attributed to an old molar stump in the lower jaw, but I could find no tenderness or swelling of the gum, all the thickening being in the cheek; thirty-six hours later an oval grey dry patch, of the area of a crown piece, appeared during the night midway between the angle of the mouth and the angle of the jaw, sharply demarcated from the surrounding tissues. The slough is now slowly separating, and bids fair to leave, if patient lives, a hole extending down to the jaw. Treated by hot perchloride dressings locally, stimulants, feeding and opium, he did fairly well until this morning, when an intractable diarrhœa, with low muttering delirium, set in, and I fear the end is not far off. The process in both cases was absolutely painless. Only 20 cases of typhoid gangrene of penis have been recorded; of the face, I believe the number is even less, but I have not the statistics at hand (Keane in his collected cases gives 47 of face, neck and trunk taken together).

Perforation.—I have had 23 cases of perforation, in all of which, I am sorry to say, we had the evidence of necropsies to bear out diagnosis. In six of these cases there was no evidence of perforation during life, but the patients were in such a hopeless condition from toxæmia that the symptoms were masked (*vide* Osler's valuable monograph on Typhoid Fever, in his "Practice of Medicine"). In the other 17 cases the symptoms were sudden and unmistakable. In 16 of

these latter I saw the cases too late to render operation admissible; in one, seen within an hour, Lieut. Smith, R.A.M.C. (whose invaluable services as operating surgeon I have previously alluded to) performed an abdominal section, sewed the rent—about the size of a tintack's head, in the ileum close to cæcum—with Lembert's suture, and washed out the abdominal cavity thoroughly. Patient, however, never rallied, and sank three hours after operation. I am now, however, convinced that operative interference is the only chance in these cases, an opinion in which my colleagues concur, and we have decided to operate in all cases seen early enough in future.

In all 23 cases the perforation was found within the last 18 inches of the ileum, and varied in size from a pin-point to a three-penny piece.

Local Neuritis.—*Foot-drop* is excessively common, both as a complication and as a sequela. In two cases there was agonising tenderness in the left calf; Osler states he has only seen two cases of this, which he attributes to myositis. Another common complication is "tender toes"—acute tenderness of the under surfaces of the toes, without ascertainable cause—first described by Handford.

Parotitis.—I have only had one case, occurring in the third week of a medium case. The swelling very slowly subsided without suppurating, and the patient recovered. This is generally regarded as a very fatal complication. Keane has collected seventy-five recorded cases.

Thrombosis.—I have had twelve cases, all affecting the crural veins, and all but one, curiously enough, the left. All twelve cases recovered.

Lung Complications.—Bronchitis and pneumonia have not been common, notwithstanding the cold frosty nights and the sudden fall in temperature at sundown; a fact that speaks well for the treatment of enteric cases under canvas.

Liver and Spleen Complications.—Enlargement of the liver is of course very common. I alluded to the case of *cholecystitis* in my last. The *spleen* has almost invariably been enlarged.

Convulsions.—In one case patient somewhat suddenly fell into violent convulsions two days after admission and died within a few hours. He had had headache and photophobia since admission, but the temperature was never above 103°. No evidence of meningitis was found after death.

I have seen no *renal complications* beyond transient albuminuria; in no case was death directly due to kidney mischief.

Hæmorrhages from Bowel.—Profuse hæmorrhages are very common and nearly always mean a fatal issue.

ENTERIC FEVER STATISTICS.

As I have now had over 1,000 cases under my care, a few statistical deductions may fairly be drawn, without in any way anticipating the elaborate tables which will be published in the future by the Army Medical Department.

(a) *Mortality.*—Of my 1,000 cases, 123 died; a mortality of 12.3 per cent. This compares very favourably with the statistics of other armies even in peace time; in civil hospital practice the mortality

often exceeds this. "The Metropolitan Fever Hospitals still shew a high rate of mortality—about 17 per cent.; and Dreschfield gives 17·18 per cent. as the death-rate in the Monsall Fever Hospital for the ten years ending in 1894. . . . In the United States Army for ten years, to 1896, there was an average annual prevalence of 138·5 cases, with mortality of 19·2 per cent." (Osler). The comparative lowness of the mortality is mainly due, I believe, to the treatment under canvas; this, during the day-time at all events, when the sides of the marquees are looped up, practically amounts to an open-air treatment. My treatment has been most commonplace, and may be summed up in few words—dieting, nursing, treatment of symptoms. I am unable to add a panacea in the form of special drug to the numerous other panaceas in existence.

(b) *Invaliding*.—Of this we cannot furnish any idea here, as the invaliding board is held at Cape Town. In any case the percentage of invaliding would be apt to mislead the civil medical public, as all men are promptly invalided home who are not likely to be efficient within a reasonable time.

(c) *Perforations*.—Twenty-three of the 1,000 died of perforation. A case percentage of 2·3, a mortality percentage of over eighteen. In Osler's 685 cases thirty-four (4·96 case per cent.) died of perforation.

(d) *Gangrene*.—Three cases; ·3 per cent.

(e) *Hæmorrhages*.—Hæmorrhages occurred in 109 cases, ninety-three of which died; 10·9 case per cent.; present in over 75 per cent. of the fatal cases, though I by no means assert that it was responsible for death in all these cases.

(f) *Thrombosis*.—Twelve cases; 1·2 per cent.

The statistics as regards inoculation will be awaited with intense curiosity by the whole medical world. I am unable to give these statistics as regards patients admitted to No. 9 General Hospital, as the men's documents do not accompany them in the field, and their statements—when not too ill to make any—are unreliable. I feel bound, however, again to have the courage of my convictions and reiterate my personal belief, viz., that inoculation, unless repeated, is useless.

DYSENTERY.

True dysentery has, I regret to say, now made its appearance. It was too much to expect that this curse of armies in the field would long remain absent. I was one of the first victims myself, being laid low shortly after posting my last communication to the JOURNAL, with a smart catarrhal attack lasting ten days. As I had on first arrival suffered from the ordinary endemic colitis of the country, I can speak feelingly of the essential difference between the two. In the former there are perhaps half-a-dozen loose blood-stained stools in the twenty-four hours, passed without straining, tormina or tenesmus, and the ailment runs its course in three or four days, leaving the general health unimpaired. In the latter, speaking for the moment only of the acute catarrhal form, there are twenty to sixty motions in twenty-four hours, most simply blood and mucus, accompanied by almost intolerable tenesmus; the disease lasts at least eight days, and leaves legacies in the shape of emaciation, debility and general depression. So far dysentery has

occurred only in the sporadic form. Some forty cases have been admitted here within the past three weeks, of which twelve have been under my own care. Of these twelve cases, eight (catarrhal) recovered sufficiently to be sent down the line for change of air, one (ulcerative) died, and three (one catarrhal and two ulcerative) remain in hospital. My microscope only arrived five days ago, so that I have only been able to examine the stools in four cases, the three now in hospital and the fatal case. In the three former nothing exceptional could be detected. The latter case is of great interest for more than one reason, so I shall relate it in some detail.

Pte. A., aged 21, was admitted to hospital on May 28, suffering from acute dysentery contracted at De Wet River. He told me he had been passing bloody stools for ten days previously, off and on, but had "stuck it out" until his sufferings became too great. He was very pale and thin on admission, with drawn anxious face; the desire to go to stool was practically incessant; griping and tenesmus almost unbearable; stools were composed almost entirely of dirty yellow mucus and pure blood. With rest in bed, milk and chicken tea diet, and a grain each of ipecac., opium, and calomel twice daily, he improved considerably, and on June 3 was passing thin, feculent slightly blood-stained stools with only slight tenesmus. The improvement continued, and on June 6 he passed only nine motions of the same character in the twenty-four hours. On the 7th inst. he became suddenly worse, tenesmus and constant desire to go to stool returned, and now the motions were most offensive, containing greyish shreds as well as blood and mucus. On the 10th there was an apparent improvement; the motions lessened in number, though of the same character. On the night of the 12th, however, he fell suddenly into a state of collapse and died an hour later. Drug treatment on and after the 7th consisted of the conventional large doses of ipecac. On the 11th I first examined the stools microscopically, preparing four slides from the mucus, as Manson recommends. In one slide I noticed two large oval bodies, which, on turning the 4th lens, I suspected to be *Amœba coli*. I placed a piece of sheet tin, with a hole cut in it, under the slide, and heated the end with a spirit-lamp; suspicion then became a certainty, as these bodies soon displayed the familiar movement, sending out protrusions and, so to speak, following into the protrusion. (I must here express my indebtedness to my friend and teacher, Dr. Manson, for the many opportunities he has afforded me of observing living amœbæ in liver-abscess pus.) I believe amœbæ could also have been found in the other slides had I made a more careful examination. The pressure of other duties prevented me making further microscopical examination during life.

At the necropsy, seven hours after death, the peritoneal cavity was found full of a reddish stinking fluid containing grey shreds and small blood-clots, and, on closer examination, a perforation the size of a threepenny piece was seen in the descending colon about midway between the flexures. On opening up the gut the whole descending colon and sigmoid flexure were found riddled with ulcers separated by islands and bridges of mucous membrane; the one which had perforated, unlike the others, was comparatively small and regular in shape, punched out right down to, and through, in one spot, the serous coat. Several of the other ulcers had gone through the muscular coat. Ulcers, but not to the same extent or depth, were also found in the transverse and ascending colons, and in the cæcum. From one of the ulcers in the sigmoid flexure, taken quite at random, I made three slides—two from its sloughy surface, one from the side. In the one taken from the tissues at the side I found three amœbæ; the size, shape, and general appearance were unmistakable, but they declined to exhibit the characteristic movement on the warm stage. I presume I had waited too long before making the necropsy. A careful search failed to reveal any in the other slides, but it is difficult to explore a slide systematically without a movable stage.

The points of interest in this case are three.

(1) *Presence of amœba in the stools*.—I believe amœbæ are rare in the stools of acute cases like this;

at all events, the "amoebic dysentery" described by Councilman and Lafleur is a very different type of disease. "As distinguished from other forms of the disease, its principal clinical characteristics are said to be chronicity, relapses alternating with periods of comparative quiescence, great liability to the formation of abscess of the liver and the presence of amoebæ in the stools" (Manson, "Tropical Diseases," 1896).

(2) *Presence of amoebæ in tissues round ulcer.*—I think this, or rather its detection, is also unusual; possibly, however, the amoeba has not been much searched for here, except by Kartulis, Councilman and Lafleur. Its presence in the *margin* of the ulcer, and its absence in the slough, is very characteristic of the amoeba; in liver abscess the amoeba is often absent from pus escaping at the time of operation, but appears in numbers four, five, or nine days later, "indicating that the habitat of the parasite is not so much the pus occupying the general abscess cavity as that immediately in contact with the wall and the breaking-down tissues themselves" (Manson, *op. cit.*).

Some three years ago Dr. Manson demonstrated the *Amoebæ coli* to rise from the pus of a liver abscess, and about the same time I remember reading the accounts of "amoebic dysentery" by Councilman and Lafleur. I recollect then feeling convinced, from the mere fact of *seeing* the amoeba on a warm slide, that the *Amoebæ coli* is the *causa causans* of true dysentery and of that peculiar necrosis of liver tissue misnamed "abscess"—a lesion just as much a part of true dysentery as diphtheritic paralysis is of diphtheria. Now that I have seen the amoeba in stools and in the margin of an ulcer I am more than ever convinced. The opponents of the amoebæ theory of dysentery make a great deal of the fact that the *Amoebæ* cannot be found in the stools in many cases; and even the American authors referred to attribute only the relapsing form of dysentery to it. May not its frequent absence from dysenteric stools be accounted for by its true habitat being the tissues surrounding the ulcers, the portal circulation, and ultimately the liver? I am quite aware that generalising from a single case must seem absurd to many, though those who have *seen* the *Amoeba coli* in stools and liver abscess will hardly think so. I can only say I shall continue to examine carefully every case I see and to keep careful records of necropsies—though fatal cases will, I hope, be few in this campaign—and will not hesitate to publish the results even if they appear to negative the amoeba theory. I hope to elicit Dr. Manson's present belief on the subject by this very crude and hastily written paper; if so, it will have served at least one useful purpose.

(3) *Perforation as the Immediate Cause of Death.*—I believe perforation to be a rare accident in dysentery. During the four years I spent in China I never saw a case. I have only two books to consult out here: Manson's "Tropical Medicine," and Osler's "Practice of Medicine." Manson barely alludes to perforation as an occasional cause of death (page 293); Osler mentions that "in 108 autopsies collected by Woodward perforation occurred in eleven."

DRUG TREATMENT OF DYSENTERY.

My colleagues and I have found the classic large dose of ipecacuanha absolutely useless. When I was

in China I gave ipecacuanha to my dysentery cases because everybody else did, though to the last I was never quite satisfied it had any good effect. I have never been in India, where we are told it acts like a charm. In my own cases sulphate of magnesia has also been a failure, increasing the patients' sufferings and not aborting the disease. At present I rely on absolute rest, support of bowels by binders, and ipecacuanha, opium, and calomel in doses of a grain each three times daily; I fancy the opium accounts for any good this drug combination does. I am more and more inclined to believe that patients would do better without any drug except perhaps a little opium at night-time to ensure a few hours' rest from tormina and tenesmus. I have had no opportunity of trying "Monsonia ovata," about which our Cape *confrères* wax enthusiastic.

MALARIAL (?) KERATITIS.

A young gentleman-trooper in Lumsden's Horse was admitted about three weeks ago to the surgical division of the hospital for a trivial bullet wound of the arm. Two days after admission he complained of photophobia and pain in the left eye, and Mr. Smith, under whose care he was, asked me to see him. On examination I found a very beautiful example of dendritic keratitis; a narrow wavy groove ran almost perpendicularly down to the centre of the cornea, and from this, thread-like grooves ran up and out, and up and in, "like the skeleton of veins in a lanceolate leaf." The cornea was much less sensitive than the other; there was a good deal of tenderness along the supra-orbital nerves. I was struck with the close resemblance of the case to those cases of "malarial keratitis" described by Kipp, Van Millingen, and others,¹ and enquired into the history. He had gone out to Assam three years before when only 20, and engaged in tea-planting, remaining in Assam till the war broke out, when he came to South Africa. While in Assam he suffered a good deal from malarial fever; he remembers the left eye being "sore" for several days on two occasions when he was laid up with fever. From his description the fever seems to have been a malignant quotidian; he had had no fever in South Africa and had been quite well during his seven months here till now. I instilled atropine, applied a pressure bandage, and gave him 5 grains of quinine thrice daily. Unfortunately we had to send him down the line four days later; before he left the keratitis had distinctly improved. I took four slides from his blood, which I examined three days ago without finding crescents or other evidence of malaria. I did not think it justifiable to examine a scraping of the ulcerated cornea.

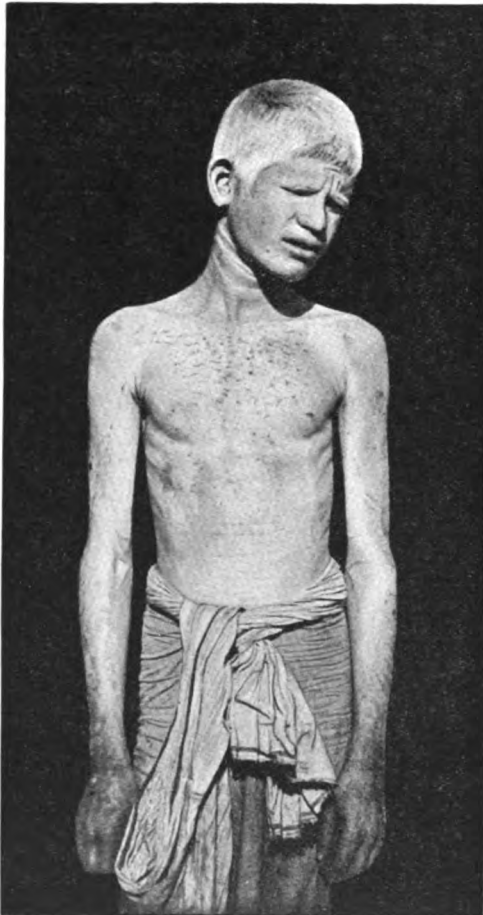
Quite recently (*Chicago Ophthalmic Record*, 1899) Dr. Ellett, of Tennessee, published a series of ten cases of malarial keratitis in which he found the plasmodium in the blood but not in scrapings; the disease was monocular and the corneal lesions similar to the above.

¹ Those interested in tropical ophthalmology will find a bibliography and full discussion of the subject in my paper, "Further Contributions to the Study of Malarial Eye Affections," read at the 1899 meeting of the British Medical Association.—M. T. Y.

A NOTE ON ALBINOS.

By FREDERICK PEARSE, F.R.C.S.Eng.

THE accompanying photograph is one of the younger of two brothers, both albinos. This freak of nature causes such a remarkable appearance amongst dark races like those of the natives of India that I thought it was worth illustrating. Mark the intolerance to light shown in the screwing up of the eyes. This is not because the boy was in the sunshine, but is a constant feature. The skin is white with the exception of the hands and lower part of the forearms, which



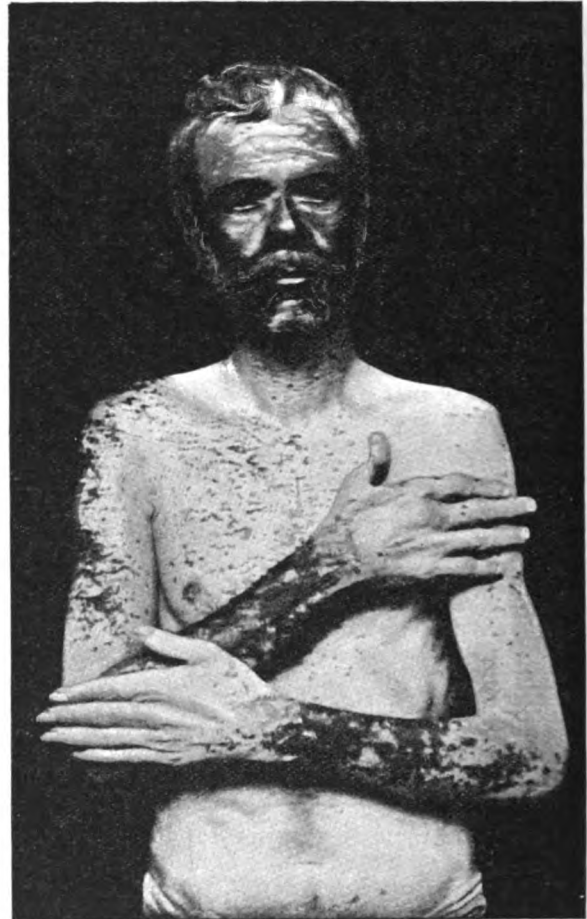
Albino: a native of India. Illustration of a case sent by Frederick Pearse, F.R.C.S., Calcutta.

showed some brownish colouration. The hair is white and the eyes are pink. A few small patches of pigmentation are distributed over his chest. His brother is similarly affected. There is no other case known in the family, either on the father's or mother's side. The other children are like ordinary natives. In Calcutta are to be seen several of these anomalies of all ages; one man I have seen has attained at least 50 years of age. There are said to be numerous cases in a village up country, about which I am making further enquiries.

LEUCODERMA IN INDIAN NATIVES.

By FREDERICK PEARSE, F.R.C.S.Eng.

THE accompanying photograph illustrates a common disease amongst the natives of India. Irregular loss of pigment forming piebald areas of skin occurs on all parts of the body. In this particular case the appearance is most marked on the man's face and arms. On the rest of his body pigment has almost entirely disappeared. He is in good health. The discolouration commenced about fifteen years ago, and has gradually progressed. No other members of his family



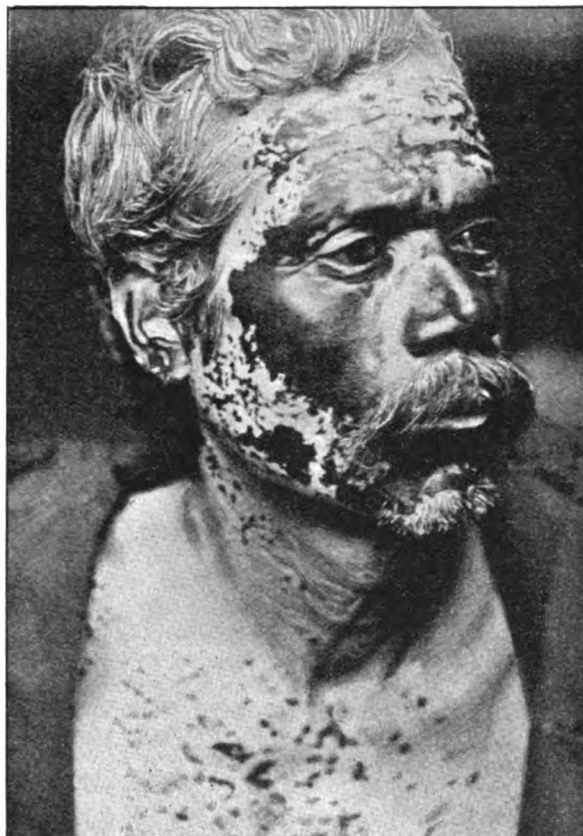
Leucoderma in a native of India. Illustration of case sent by Frederick Pearse, F.R.C.S., Calcutta.

are affected, and he suffers no pain or other form of discomfort. The disease is popularly called white leprosy, but has no relation whatever to the true kind. Children as well as old people are frequently to be seen having the same condition. Some of the natives lose nearly all their colour and become almost like white people, others only lose the colour in small patches. Unexposed parts of the body seem to be equally liable to be affected as those uncovered. I have no information as to whether pigment once lost is ever recovered. The hair and the eyes retain their colour.

CIRCUMSCRIBED CUTANEOUS OEDEMA IN BRAZIL.

By W. G. TOTTENHAM POSNETT, F.R.C.S.I.

QUINCKE, in 1882, described what he called "circumscribed cutaneous oedema." Since then dermatologists seem to be confused between this affection and so-called urticaria gigans. Not being a specialist on skin diseases I do not intend to discuss the difference between these cutaneous maladies, but to record observations of six cases which I saw while acting as ship's surgeon to Brazil. I am led to do so by the fact that in my reading I have never seen it men-



Leucoderma in a native of India. Illustration of case sent by Frederick Pearse, F.R.C.S., Calcutta.

tioned that this disease is prevalent in any part of the tropics.

The first case was that of a sailor who told me that, as soon as the ship arrived at Para, each voyage he got an attack of what he called "lumps all over my body." I told him to come to me the first time he saw any of these "lumps." While going up the river to Para he came and showed me his shins, on each of which was a single swelling, purplish in colour and having a tingling, burning sensation. Both were about the same size, $2\frac{1}{2}$ by $1\frac{1}{4}$ inches. The morning after arrival I woke with a tingling sensation in my left fore-arm, on examining which I found to have a swelling on it, reaching from the olecranon to the back of the wrist. There was a decided sense of

weight in the arm, but not much itching. There were also several large swellings on my left buttock.

The next morning the back of my right hand was so swollen that I was "hide bound" and could not hold my razor. This passed off in about five hours.

One of the saloon passengers was attacked the next day. He was very ill, shivering, vomiting, diarrhoea and a temperature ranging from 101° to 103° . The general symptoms appeared a few hours before the "rash," and subsided to a certain extent when it became developed. This case lasted twenty-three days. The patient had resided in Brazil for some years. The other cases presented nothing peculiar; one occurred on the voyage up the Amazon, the other two while we were lying in the Rio Negro at Manaos.

Taking all the cases together the following were the main points noticed: the onset was always sudden, accompanied by slight malaise, the swellings appearing in the morning, some persisted throughout the day, others for two or three days, while some disappeared in a few hours. In none of the cases did swellings appear in the day time. Itchiness was very slight, the sensations complained of being of a tingling and burning description.

With regard to the individual swellings, a surrounding blush was seldom seen well marked, the cedematous portion of the skin was paler than the healthy, unless it had been rubbed or scratched, when it became a deep purplish red; pitting on pressure was observed, to a slight extent, in some of the cases. The situations attacked were the dorsum of feet and hands, shins, back and front of the thighs, buttocks, back of shoulder and dorsal aspect of the forearms. The face was in none of the cases the seat of swelling.

The swellings were always of an oval shape, never circular, and I noticed that in the limbs the long axis of the swelling was always in the long axis of the part, those on the buttocks and shoulders had a direction downwards and outwards, seldom inwards. This peculiarity of direction I have not seen noticed in any of the text books on dermatology which I have consulted.

All the cases occurred among Englishmen, and on enquiring into the subject I found that it is a comparatively common affection among the English in Brazil, but rarely attacks Portuguese or Brazilians. Diet played no part in these cases, the cause I could not trace. The duration of the cases was variable; the first lasted but one day; my own, untreated for observation, four days; the others, five, three, seven, and twenty-three days.

Treatment consisted in a mixture of pot. bicarb., mag. carb. and mag. sulph., which in the first case did undoubted service, as it was the first voyage for five that the sailor had not an attack lasting from one to two weeks. In the other cases I think treatment had little to do with recovery. In the passenger's case sod. sulph. seemed to be of most benefit. Local applications I consider useless.

I was told by a medical man that he had seen suppuration of the swellings occur while at Santos, and that it is not uncommon in the Argentine. In all probability, if looked for, this disease would be found to be widely prevalent in South America.

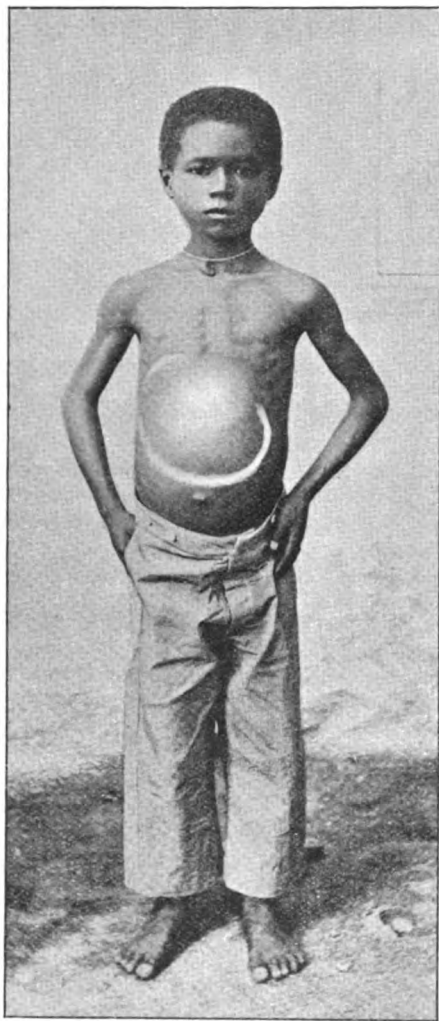
CLINICAL PEDIATRICA.

A CASE OF CIRRHOSIS OF HANOT.

By Dr. FERNANDES FIGUEIRA.

Ex-assistant Surgeon of the Clinical Pediatrica of the Faculty of Rio de Janeiro.

ON January 23, 1898, there came for examination a boy Octavio, 10 years of age, a mulatto employed at a farm. The father of Octavio stated that during two years his son was laid up four times with severe attacks of jaundice, from which he appeared to partially recover but never entirely. At an early period



Cirrhosis of Hanot. Illustration of a case reported by Dr. Fernandes Figueira, Rio de Janeiro.

The white line indicates the circumference of the liver. The original photograph, taken in a very bad light, did not show the size of the abdomen.

his mother died from tuberculosis of the lungs, leaving this only son, at the time three years of age; up to that time he was always healthy. When from five to six years of age he on several occasions indulged in drinking brandy even to intoxication; he, however, lost this bad habit, thanks to paternal correction. The present disease, according to the only trustworthy

information, showed itself months after he had drunk by mistake coffee mixed with sweet calomel (so his father states). The misadventure, however, according to the statement of the same informant, was not followed by any phenomenal or unhealthy consequences, which overthrows the necessity for such information.

The examination showed: Height, 120 centimetres; dentition normal; no symptoms of syphilis, of arthritis, or any suspicion of tuberculosis; thoracic organs perfect; the abdomen is distended without ascites; the thoracic perimeter 66 centimetres; abdominal circumference at the level of the umbilicus 67 centimetres, at the level of the crest of the ilium, 57 centimetres. One sees by these measurements that the thorax in the submamillary section shows almost the same perimeter as the abdomen at the umbilical level, becoming narrower in a remarkable degree from this point downwards. The belly is enlarged and hard; this enlargement is owing to the liver and spleen—the first normal as to its upper limits, measuring below the costal border in the middle line 15 centimetres, in the right nipple 6 centimetres, in the left $4\frac{1}{2}$ centimetres, and extending but slightly above the costal border. The spleen measured in the axillary line 8 centimetres below the costal border, having the same inferior maximum level as the liver, from which it is separated, in the anterior aspect of the abdomen, by a distinct space equal to 4 centimetres. Both these organs are hard and but slightly sensitive to pressure, the lower border easily perceptible and without protuberances. There were no enlarged abdominal veins nor hepatic murmur. Eyes showed jaundiced tint. Slight enlargement of the right knee joint in which the patient complains of feeling slight pains. Stools not pale coloured. Tongue furred. Mental state perfect. Appetite good. Urine acid, yellow, with a density of 1,014, without albumen. Reaction clearly that of Gmelin; reaction also of Pettenkofer. Urobiline clear to the spectroscope.

Regimen.—Pills of podophyllin and rhubarb, inrubeba wine with iodide of potassium. Milk diet.

Feb. 2.—The disease appeared stationary, the regimen not having been kept up.

Regimen.—Inrubeba wine with boldo and pichi. Friction with Neapolitan mercurial ointment in the hepatic region every three days.

April 6.—Pains in the left knee, which is fairly increased in size; lichenoid eruption on the forehead. The spleen retaining, at the anterior aspect of the abdomen, the same parallel in relation to the left side of the liver, but at a distance of $2\frac{1}{2}$ and not 4 centimetres as previously. The abdominal perimeter at the navel is enlarged by 6 centimetres. Slight dropsy.

Regimen.—Sabricus australis wine, oxymel of squills, nitrate of potash.

May 14.—Stationary.

Regimen.—Calomel and scammony taken every morning, with exclusive milk diet.

May 18.—The dropsy disappeared, as also the swelling of the knee; Octavio feels better.

Regimen.—Oxymel of squills, syrup of inrubeba, and sabricus australis.

June 5.—Octavio is much worse; œdema of the extremities reaching up to the knees; dyspnoea, weak

pulse, dropsy. Drastic purgative, decoction of elder bark. Epistaxis in the night.

June 6.—The difficulty of breathing increased; Octavio is not able to leave his bed; the liver is slightly painful on being pressed; the emaciation is remarkable.

With the intention of alleviating the patient I tapped him, which only relieved him of barely a litre; a very little bilious liquid.

June 7.—The swelling diminished. I tried opotherapy, making the patient take daily 60 grams of the infusion from the fresh liver of the pig.

With this treatment and the diminished swelling Octavio felt better for the space of six days, but the objectionable signs still remained. At the termination of this period, eluding the vigilance of those who had charge of him, the patient consumed an abundance of oranges and indigestible nutriment, violent diarrhoea and sickness supervening, followed at once by adynamic phenomena, which in twenty-four hours proved fatal.

The study of the present observations offers various points for consideration. They treat of a case of jaundice freely metapigmentary, as proved by its surroundings: the reaction of Pottenkofer, the special coloration, together with the natural colour of the stools, and the spectroscopic examination; all point to degeneration of the hepatic cells. Nevertheless, during six months, and up to even four months before death, the urine showed the reaction of Gmelin.

Then little by little the hepatic cells were invaded until completely unfit to sustain life.

There existed a cirrhosis, the cause of the jaundice, and the spleno-hepatic enlargement which does not exclusively belong to the disease of Hanot in children, as erroneously thought, but which is observed in other types of infantile cirrheses.

Excluding the syphilitic tendency there is no sign of marked diathesis, nor lobulation in the liver, nor dropsy, nor enlarged abdominal veins, so that the jaundice, seldom found in it, was in this case an extraneous symptom.

The absence of damage to the cardiac orifices and pulmonary tissues, the remarkable ascites, and the continued jaundice, besides the symptoms of less importance, proclaim at first sight the exclusion of cirrhosis cardio-tuberculosis.

Much less can one build up the hypothesis of a cirrhosis of Lorennia. There remains, however, paludism and the hypertrophic cirrhosis of Hanot.

Signs of paludism were altogether wanting in this case.

The signs of hypertrophic alcoholic cirrhosis are also absent.

There remains Hanot's disease—our diagnosis—as it is described in its first stage by Gilbert and Fournier. In the composition of the urine, in the articular symptoms, the lichenoid eruption, in the hemorrhage, in the spleno-hepatic enlargement and even in the obscurity of the cause of the disease, the case is typical.

The physical observations certainly lack the confirmation of an autopsy—a confirmation well-nigh Utopian in this country. Nevertheless it appears that to a dozen cases of the cirrhosis of Hanot in

infancy studied in Europe, may be added the present case, for the first time noted in Brazil, a tropical country, and in a half-caste individual—a circumstance which perhaps may add some interest to these imperfect observations.

[This is a case in all probability of biliary cirrhosis, with a secondary portal cirrhosis supervening. The portal cirrhosis would account for the ascites, which is never present in typical cases of Hanot's cirrhosis.—J. C.]

ANCHYLOSTOMIASIS IN THE LEEWARD ISLANDS.

By WM. M. McDONALD.

Acting Medical Superintendent, Holberton Institution, Antigua.

DR. NICHOLS seems to have written a paper on his experience in Dominica and on my despatch to the Government. He does not mention the state of affairs in Montserrat, Nevis and St. Kitts.

Dr. Nichols seems to think that I made a report to the Government on cases in which I had found the parasite, but in which there were no symptoms of ancylostomiasis. If Dr. Nichols had taken the trouble to read my report correctly, he would have seen that I drew the attention of the Government to this disease for the following four reasons:—

- (1) That up to that time ancylostomiasis was not recognised and not treated in Antigua.
- (2) Its great prevalence throughout Antigua.
- (3) The severity of its symptoms in all the cases I reported.

(4) Its response to treatment, if attended to early. I do not examine the stools of all patients that come into the Holberton Institution; if I did no doubt I should find the parasite in the majority of cases. In those cases that I reported to the Government the stools were examined on account of the marked symptoms of ancylostomiasis. So marked were the symptoms that in most of the cases the nurses noted the nature of the disease at once and put the cases by themselves.

Dr. Nichols is ignorant of the condition of affairs in Antigua. He argues safely from his experience in Dominica, where he confesses that the chances of infection are much less than in Antigua. He further says that in a number of cases in "Roseau Hospital," in only a few instances has it been possible to class the cases as pure ancylostomiasis. But knowledge of ancylostomiasis limited to Dominica does not prevent Dr. Nichols from criticising my paper, and from assuring the Government that I have overstated the importance of ancylostomiasis in Antigua, and that they need not be alarmed.

In an island like Dominica one would hardly expect to find many cases of ancylostomiasis, for in that beautiful island there is a sparse population, and there is an abundant supply of pure running water.

In Antigua the conditions are exactly opposite, for here the population is much greater, and the water supply in the country is of the worst—only dirty pond water; all the cane fields are open latrines. During heavy rains the wash from the cane fields finds its

way into these ponds that supply drinking water to the natives. Could there be more favourable conditions for spreading all kinds of disease?

My despatch to the Government was written, I think, on 40 cases that came into hospital in four months. Now that a year has passed away I do not alter in the slightest my opinion of this disease in Antigua. I still call it a "terrible disease," and I still say that it is "sapping the life and energy out of the labouring population."

In the last thirteen months there have been 148 cases of "anchylostomiasis" in the Holberton Hospital, and of this number 34 have died in spite of vigorous treatment.

All these 148 cases showed marked symptoms of anchylostomiasis—profound anæmia, debility, palpitation and attacks of syncope; they had all been unable to do any work for from six to twelve months, although they were all in the prime of life, their ages ranging from 16 to 50 years. They had been living on either the charity of their friends and relations, or on the poor relief given by the Government.

I have specially studied all these cases, and taken careful notes of their past history and of their symptoms, and I have been careful to diagnose them from Bright's disease, malaria and beri-beri. In none of these cases has there been any albumin in the urine, or has there been a single case of enlarged spleen or any paralysis.

It must be remembered that these 148 cases do not nearly represent the full state of affairs in the whole of Antigua, for there must be many others about the country suffering from this disease.

I think this disease can justly be called "terrible" in Antigua. Think what has been going on here for years. It can be said that nearly all these 148 cases would have died by now if they had not been treated; imagine what this means if you carry this back for the last fifty years.

These cases were not recognised and were not treated, they slowly went to their graves, many being paupers, all being dependent on charity, and all being a source of the greatest danger to their neighbours, when one considers the conditions in which they live. It is not surprising, then, that this disease has reached such alarming proportions in Antigua, for here you have an ideal state for the spread of disease; the dirty pond water for drinking purposes; the cane fields, open latrines, where the natives deposit their fæces without restriction; this has been going on for ages, unchecked, nothing done to improve the health of the natives, or treat the disease, or improve the unsanitary surroundings of these naturally filthy people.

"Anchylostomiasis" is an *exceedingly chronic disease* in most cases. It takes years at least for the anchylostoma to reduce an ordinarily robust person to a state of "anchylostomiasis." Is it not natural therefore that there must be many persons harbouring this parasite who are not yet reduced to a true state of "anchylostomiasis"? There are numerous ova of the anchylostoma found in their fæces, but they are in apparently robust health.

Dr. Nichols says that "in the enthusiasm of working in a new field many observers have over-estimated

the harmfulness of a moderate infection." I say most definitely that the harmfulness of a moderate infection *cannot be over-estimated*.

Imagine what even a moderate infection would mean to an individual suffering from chronic starvation (a very common complaint in Antigua at present) or typhoid fever, Bright's disease, dysentery or malaria. As Manson says: "in these countries there are many who live just on the borderland between health and disease; to such the anchylostoma may prove the last straw."

Dr. Nichols says that a moderate abstraction of blood in plethoric individuals may not cause deterioration of health; no one will deny that, but where are these plethoric individuals in the tropics?

Thornhill (1895) regards its ravages as far more serious than those of cholera; this, not on account of the number of deaths it causes directly, but on account of the vast numbers affected, the chronic nature of the disease and the aggregate mortality, direct and especially indirect, for which it is to be held responsible.

Dr. W. S. Law, in the *British Guiana Med. Journal*, 1897, says: "Anyone who has had experience of medical work in this country knows how many coolies drift every year into our almshouses, the early stages of whose decline was merely one of slowly increasing anæmia. Suppose even 50 per cent. of these were really, at the beginning, cases of anchylostomiasis—and this supposition is not fanciful—it would be easy to calculate how much would have been saved to the colony had their condition been correctly diagnosed at the start."

I think the cases that harbour the parasites, and that do not show symptoms, are just as important as those suffering from anchylostomiasis, for a large proportion of those that show marked symptoms of anchylostomiasis are "past praying for," they go downhill whatever the treatment; now on the other hand, those that harbour the parasite without any definite symptoms, with vigorous treatment are prevented from becoming chronic invalids, a nuisance to themselves and their relations, a burden to the State, a serious loss to their employer, and a source of great danger to their neighbours.

However lightly Dr. Nichols may regard "anchylostomiasis" in Dominica, I can only regard it, after thirteen months' experience, as a "terrible disease" in Antigua; and no one can deny that anyhow in thirteen months anchylostomiasis in Antigua has "sapped the energy out of 148 persons in the prime of life, and also sapped the life out of 34 unfortunate victims."

A SANITARY CORPS FOR THE BRITISH ARMY.

By SAMUEL OSBORN, F.R.C.S.

(From experiences gained during the South African Campaign).

THE extraordinary prevalence of enteric fever and the fearful mortality therefrom which has befallen our troops in the present South African campaign demands that some precautions, more than we at present adopt, should be undertaken in the future.

When the final statistics are stated I am quite certain that the mortality from this fearful scourge will be found to be far in excess of those who died from actual wounds received upon the battle field, and that the percentage of deaths will be far in excess of the proportion of deaths occurring from the same disease in our general hospitals.

I can fully endorse the remark of Dr. Conan Doyle that the outbreak of enteric fever among the troops in South Africa was a calamity, the magnitude of which had not been foreseen, and which has never before been equalled.

It was on May 29 that I entered Kroonstad in the early morning with Lord Methuen's column and paid a visit to the sick in that town. In a letter written that very evening I describe my visit in these words: "The village is the abomination of desolation, nothing worth buying to be got, no eatables at all, and the greater number of the shops shut up. Every house and both hotels are hospitals with enteric fever cases. I went into the chapel, and there were ninety cases being treated there, some of the poor fellows undoubtedly dying, and the smell in there was something horrible. In the bell tents in the chapel grounds were about 200 more cases. This is not counting those scattered about the town in almost every house. Lord Dunvargen and myself went to the Grand Hotel, hoping to get something to eat and drink, and we were told that nothing could be supplied to us, as this was a hospital also. At this time there are in Bloemfontein alone 5,000 sick in the hospitals, of which over 4,000 are enteric fever cases." This number of enteric fever cases did not include any of those being nursed in other military hospitals in other places scattered over the country, to say nothing of the numerous cases dropped down in out-of-the-way places in many towns and villages, where, in the course of the rapid advance of our troops, we were compelled to leave them in town halls, schoolrooms, and private houses, temporarily transformed into base hospitals.

My remarks, however, are not as to what has been, or how this unforeseen and unprecedented crisis was met, but rather as to how in the future such an outbreak could be mitigated, if not obviated. It is early yet to say much about the benefits of anti-typhoid inoculation, as regards which I am personally somewhat sceptical. Several soldiers going out on my steamer to South Africa were so treated, and the after effects of the inoculations were in some instances so severe as to cause many others to refuse to have it done. Some excellent statistics as to the immunity obtained by inoculation have been compiled by Colonel E. Townsend, R.A.M.C., of those cases occurring down at the Modder River, and which when published will throw much light on this subject, and, I believe, in its favour.

What I am more especially desirous of advocating is the formation of a "*corps sanitaire*" in our army, like we find in all Continental armies. Such a corps would undertake the selection of camps, the supply of pure water, the erection, proper closing and subsequent marking of old latrines, the use of disinfectants, and the choice of proper buildings in towns as hospitals, where the sanitary arrangements are often primitive if not altogether absent, &c.; in fact, all the sanitary

requirements appertaining to a large army in the field. It is in this direction, to my mind, that there appears to lie the room for alteration and improvement.

One column on the march is often called upon to occupy the same ground as one that recently preceded it. The latrines may have been properly disinfected and subsequently filled up, but allowing this to be the case, no distinguishing mark of their situation is placed to guide and warn the second column from occupying unhealthy ground, and consequently mess and other tents may be pitched upon or in the near proximity to most undesirable spots.

Again, the soldiers are not sufficiently careful to use solely those places so arranged for their use, and the camping ground becomes defiled. I know that this was sometimes the case, as in camp orders, on one occasion I remember, an admonition occurred that one column had left their old camping ground in a very unsatisfactory condition to the annoyance and possible danger of the column following it.

Dead horses, oxen and mules marking the line of march and lying by the roadside did not improve the sanitary condition of affairs; and the peculiarly dry character of the atmosphere does not allow these dead bodies to putrify, but rather to dry up or mummify, and the dried particles are blown about with the dust of the veldt.

Again, the soldiers on the march halt every hour, when they fall out anywhere for nature's purposes, and the excreta here, in the same way becoming dry, is blown about by the wind, and carried in the dust becomes inhaled or swallowed by the men following subsequently the same route. And if, as I know to have been the case, any of these soldiers had premonitory signs of enteric fever, the result and spread of disease is obvious; because many a soldier will suffer for days before he will give in and report himself sick, as he has no desire to be left behind or sent down to a base hospital, and all chance of taking further part in the campaign be put an end to.

The dust from an advancing column extends not only along the line, but for hundreds of feet on its leeward side and for as many feet upwards—a fact of recognition of the locality of moving troops known to many a good scout on the look-out.

That the dust was an important factor in the cause of intestinal trouble was known to many, and consequently when they could they rode their horses to windward of the column whilst on the march. I know of several instances where the dust was undoubtedly the cause of men falling sick.

That the formation of such a sanitary corps would be productive of much good I am quite certain; and if by its formation a more sanitary condition of affairs was produced for our army on active service, I believe, the terrible ravages occasioned by enteric fever, although at present to a certain extent inseparable from any campaign, would be considerably decreased if not absolutely put a stop to.

THE RECENT DISCOVERIES ON PALUDISM.

By Dr. J. GUIART.

Translated from the French by P. Falcke.

Paludism, which is still known in France under the name of intermittent fever (and as *ague* by the English, *Wechselfieber* by the Germans, and *Malaria* by the Italians), is an infectious illness of parasitical origin, caused by the propagation in the blood of a parasite belonging to the order of Protozoones. Perhaps paludism is the most common affection; it is at least the one most widely spread on the surface of the globe. It has been known from all time, and its antiquity is as undeniable as its geographical dissemination is extensive.

Geographical Distribution.—It is undeniable that, speaking generally, paludism, unknown in cold countries, increases in frequency and intensity as the equator is approached, and within tropical zones it may be said that localities exempt therefrom form the exception.

In France, paludism is endemic on certain parts of the coast of the Mediterranean Ocean (Camarque, Landes, Charantes, Vendée), and in certain centres in the interior (Lologne, Brenne, Bresse, Dombes). The shores of the Corse are almost uninhabitable owing to malarial fevers. One may also remember the ravages made by malaria in the French army and amongst the colonists at the commencement of the conquest of Algeria; but happily the scourge is less terrible now-a-days, on account of the cultivation of the soil, which has partly caused the disappearance of morasses, and has produced a marked improvement of the sanitary conditions. This, however, is unfortunately not the case in most of the French colonies; the Antilles, Cochin China, Guiana, Tonquin, Senegal, and the Congo, have an unenviable notoriety, and everyone still remembers the murderous campaign of Madagascar, where, in consequence of the inefficiency of the administration, so many thousands of brave soldiers fell victims to malaria. New Caledonia, which is almost free of this illness, is on this account considered the healthiest French colony.

Medical geography shows that damp is the most important factor in the genesis of malaria, and indeed the principal centres are situated on the shores of the great streams and lakes, and above all, in countries that are marshy or frequently flooded. Abundant rains favour the development of the illness. Taken altogether, it may be said that malaria prefers uncultivated and sparsely populated countries, while it retreats from civilization. This is really because, in rich and well-populated countries, the damming of rivers and the drainage of marshes prevent the affection from finding a centre favourable to its development.

A slight altitude, also, is unsuitable to the existence of malarial fevers. Thus at Constantine, the interior of the town is very healthy, while 100 mètres lower, on the shores of the Rummel, malaria prevails with terrifying intensity. In the same manner it has been remarked that, in an affected town, the elevated quarters are free; and even in the lower lying parts the inhabitants of the higher flats of a house are less

liable than those on the ground floor. Later on these various etiological differences will be explained.

Symptomatology.—Typical malaria is a febrile illness, which is characterised by attacks. Each of these attacks returns at intervals of 72, 48, and 24 hours, thence the names *quartan*, *tertian*, and *quotidian*, which are the principal varieties of the illness, and which will be better understood by the following table, in which 0 indicates the days without attacks:—

$$\begin{array}{cccccccc} 1 & 0 & 0 & 1 & 0 & 0 & 1 & 0 & 0 & 1 \end{array} = \text{quartan.}$$

$$\begin{array}{cccccccc} 1 & 0 & 1 & 0 & 1 & 0 & 1 & 0 & 1 \end{array} = \text{tertian.}$$

$$\begin{array}{cccccccc} 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \end{array} = \text{quotidian.}$$

Every attack is composed of a stage of shivering, a stage of heat, and a stage of perspiration. The shivering consists in such a universal and intense feeling of cold that the patient's teeth chatter, and he shivers from head to foot, and he tries to cover himself with all the wraps at hand. If, however, his temperature be taken, it is found to be several degrees above normal, and rising rapidly. After about an hour the shivering gradually abates, to make way ultimately to a sensation of intense heat. The patient throws off his wraps, headaches and vomiting are frequent, respiration is accelerated, the skin is dry and burning. The thermometer placed in the axilla registers 40°, 41°, 42° C., and sometimes even more. After three or four hours of suffering the patient is taken with profuse perspiration. The perspiration literally pours off him and soaks the bedclothes. Then the fever rapidly diminishes, and the sufferings caused by headaches, vomiting and thirst are replaced by a feeling of relief and well-being. As soon as the perspiration has ceased the patient feels quite well, and can accomplish his usual occupation. The duration of an attack is variable, but on an average lasts from six to ten hours.

This is a short summary of the principal symptoms of the classical acute attack of intermittent fever. But the stages do not always succeed each other so regularly. As a rule, the attacks have a tendency always to reappear at the same hour, but in certain cases the attack sets in every time a little earlier; the fever is then said to be "anticipated." If, however, on the other hand, the attack is an hour later it is said to be "retarded." When the crises are prolonged in such a manner that one is not finished when another commences, the attack of fever is called "subintransient." When the fever is prolonged and is simply marked from time to time by a slight fall of temperature, a slight perspiration, and a slight sensation of cold, the fever is called "remittent," but sometimes there is no remission, and then is named "continued."

In certain cases, on the other hand, the symptoms are very slight. At times the illness will only be characterised by a sensation of cold recurring periodically, followed by headaches or by a slight rise of temperature; that is the *larval* form of malaria. It must not, however, be supposed that this is the least dangerous form; the patient may be able to attend to his business with a temperature of 39° or 40° C., but

it must be remembered that certain fevers of Africa (so liable to suddenly assume a pernicious character) are of this type. The diagnosis, however, is very easy, an examination of the blood being sufficient to clear up any doubt.

As to the serious forms of malaria, which are designated *pernicious fevers*, they may be initiated by a multitude of causes—be it intercurrent maladies, such as typhoid fever, yellow fever, cholera, dysentery, &c., be it individual predispositions accentuated, say, by alcoholism or syphilis. But the parts played by these differences have hardly been analysed, so that they are forms but little understood in reality and on which we shall not enlarge for that reason.

Anatomical Pathology.—Melanæmia, in other words, the abundance of pigmented elements in the blood, and particularly in the vessels of the spleen, the liver and the brain, giving these organs a pronounced brownish tint—that is the characteristic alteration of malaria; the pigmented elements come from the destruction of the malaria parasites, which feed themselves at the expense of the hæmoglobin, thus causing the intense anæmia of malarial subjects.

THE MALARIAL PARASITE.

Twenty years ago it was a common belief that malaria, like most infectious illnesses, was produced by a bacterium, and the bacillus, which *Klebs* and *Tommasi Crudeli* described under the designation of *Bacillus malarie*, had numerous partisans. No one then suspected that one of the most extensively disseminated illnesses on the face of the globe was due to animal parasites living in the blood of man. Therefore when, in 1880, Laveran discovered the hæmatozoë of malaria in the Military Hospital of Constantine, the description he gave was received with much scepticism. But now so many observers have verified the existence of the same that no one any longer dreams of denying it. The parasite of which we are now about to speak is a micro-organism, coming under the class of protozoa, the lowest class of animal life. Most authors coincide in classing it with the sporozoa, in the order of the Cocci. Soon after its discovery it received the designation of *Plasmodium malarie* from Marchiafava and Celli.

Microscopic technicality.—Care should be taken to examine the blood of those persons suffering with malaria who have not taken quinine. The commencement of the attack is the most favorable opportunity. The examination of the fresh blood is a most interesting one, and one may then observe the movements of the parasite and even some of its transformations. It is indeed by these proceedings that Laveran was enabled to make his memorable discovery.

The tip of the finger of a malarial subject should be soaped and washed with alcohol; then with a previously heated needle the pulp of the finger is pricked. The first drop of blood is wiped off and the following ones are received on clean slides. The specimen can be examined directly by simply covering the blood on the slide with a cover glass, or the organism may be stained while living. To accomplish this one drop of methylene blue dissolved in a solution of chloride of sodium (0.75 gram. of chloride of sodium to 100 cubic centimeters of water) is dropped

at the side of the drop of blood. The cover glass is superimposed, the two liquids amalgamate, the parasites absorb the colour, which has the advantage of being non-toxic, and in a little time the parasites stained blue will be seen on the uncoloured base, when it is easy to observe their movements.

But if one wishes to study the structure of the hæmatozoön it is absolutely necessary to have fixed preparations. For this purpose each drop of blood is collected on a slide—previously disinfected and heated, but only to the extent that it is bearable to the hand. The drop of blood is deposited at the edge and with the aid of a cover glass is rapidly spread from end to end while blowing quickly in order to assist the desiccation. Care must be taken to accomplish the spreading in one effort so as not to break the elements. In this manner one is certain to have but one layer of corpuscles. The preparation is then fixed by covering it with equal parts of pure alcohol and ether and allowing it to evaporate in the fresh air. This accomplished, the preparation is fixed and has only to be stained. This can be well and quickly done by treating it for half a minute in eosin dissolved in alcohol at 60°. By this means the red blood corpuscles are stained pink. It is then washed in water, and it is treated for another half-minute by a concentrated aqueous solution of methylene blue which stains the parasites blue. It is then again washed in water, allowed to dry, and examined direct through the microscope, with the objective in immersion, or it may be dehydrated by pure alcohol, cleared by xylol and mounted.

In order to study the histological structure it would be preferable to mix eosin and methylene in equal quantities and to leave the glasses in this mixture for twenty-four hours. It is then carefully washed.

Description of the parasite.—The parasite is exhibited in the blood under various forms, which may be found in any one of four types:—

(1) *The spherical bodies.*—These are the forms most commonly observed, and they possess amœboid movements, hence the name *amœboid bodies* which is sometimes given to them. Laveran asserts that these parasites are simply fastened to the red blood corpuscles, but at the present day it is universally accepted that they are in the interior of the corpuscle itself. The smallest of these amœboid bodies sometimes exhibit one or two particles of black pigment, but in proportion to the increasing size of the parasite the grains of pigment become more numerous and are then most frequent, arranged as a peripheral wreath in the ectoplasm. In the endoplasm is observed the germ, which is difficult to stain, and which is presented as a central uncoloured space, enclosing a voluminous nucleus.

(2) *Rosetted bodies.*—These are also called *segmented bodies*, and they represent one of the methods of reproduction of the parasite. If the blood be examined at the commencement of an attack of fever a certain number of the spherical bodies are seen in which the grains of pigment have collected in the centre, while the germ itself is divided into a number of larger and smaller divisions extending to the periphery. The protoplasm also becomes segmented, and the result is a series of small round bodies or sporozoa no longer

containing pigment and which soon become free in consequence of the rupture of the blood corpuscle. These are the agents of the dissemination of the parasite in the organism; these are the agents of auto-infection.

This *rosette* form has a particular evolution in the different varieties of malarial fever. Thus the quartan is characterised by segmented bodies yielding only from six to twelve sporozoites, while the tertian is characterised by bodies in the form of daisies which give off fifteen to twenty spores. According to Italian authors, these rosette forms are due to parasites of a different species, but in France, Laveran, Labbé, and Metshnikov assert that they are simple varieties of one and the same parasite. According to the latter author, it should be with *Plasmodium malarie* as with a bacterium which divides more or less quickly, according as the means are favourable or otherwise to its development.

The same is the case if the hæmatozoon attacks a favourable organism; it will divide rapidly, and yielding more spores, the infection will take place more rapidly, hence the tertian type. But in a less favourable organism it will give off but a few spores, and infection taking place less quickly, the attack will be delayed or of the quartan type. As to the quotidian, that would be the result of the simultaneous evolution of several generations of parasites. Such is the theory of the specific unity of the parasite. At the same time we must acknowledge that lately most authors seem to come round to the opinion of the Italian investigators as to there being three distinct hæmatozoa: the *Plasmodium malarie* which gives birth to the quartan, the *P. vivax* to the tertian and the *P. præcox* to the æstivo-autumnal fever. The other forms are supposed to be secondary and produced by the simultaneous evolution of several parasites in one person.

(3) *Crescent bodies*.—These peculiar bodies are mostly observed in cases of malarial cachexia and also in the æstivo-autumnal fevers. In the blood corpuscle their development is marginal, avoiding the central portion; they are thinner, and thus have gained the name of crescents, their convex side moulding itself exactly to the circumference of the corpuscle. In their centre the agglomeration of pigments is observed, as also the nucleus, according to some authors. At this stage the parasite has destroyed nearly all the hæmoglobin of the red blood corpuscle, and that which remains can be seen in the concavity of the crescent. At last the crescent becomes free in the serum.

Thus they are observed in the blood, but their ulterior evolution seems to be produced outside the human body; they also are considered to be the encysted form, or form of resistance, of the parasite. This explains why they are so frequently met with in old cases of malaria. If a drop of fresh blood be examined under the microscope, the crescents are observed first to transform themselves into ovoid bodies, then into spherical bodies, and at last, at the end of fifteen or twenty minutes, a certain number of these spheroid forms have power to give birth to the fourth form to be now described.

(4) *Flagellated bodies*.—We have seen above that the spheroid bodies can reproduce themselves in the

blood by simple division, in a manner to originate the rosetted forms. But since the publication of Maupas' remarkable works we have become aware, that, with all these protozoa, this faculty of non-sexual multiplication becomes exhausted in the long run, and the species would become extinct did not sexual reproduction intervene, and this is precisely the object of the flagellated bodies.

If, again, a drop of blood be microscopically examined, it may be seen that certain modifications are produced in those spheroid bodies free in the blood, which proceed from the sporozoa or the crescent bodies. The nucleus divides into a certain number of chromatic particles which proceed towards the periphery and emerge from the protoplasmic mass perpendicularly to the surface, dragging out into long, extremely thin filaments. These filaments, generally four in number, are extraordinarily mobile, their movements may be compared to those of little eels which, held by their caudle end, attempt to free themselves. After flogging about for some time the flagellæ at last become detached, and being free in the blood move rapidly amidst the globules and are soon lost sight of. Laveran looks upon these flagellæ as the adult and perfect state of the hæmatozoon of malaria. Metshnikov and Simond consider that they represent the male sexual element of the parasite; a condition analogous to the formation of the microgametes of the cocci. It will be seen indeed that these represent the agent for the dissemination of the parasite outside the body, and are the agents of fructification. This serves to explain why these flagellæ do not appear in the blood till after it has left the vessels, when the blood has been exposed to the air some seconds. As to the opinion of certain authors who regard the flagellated bodies as a form of agony preceding degeneracy, it is at the present to be considered only as of purely historical interest.

By their chromatic constitution and their mobility, these flagellæ are to be considered as the male element of the parasite analogous to the spermatozoa of man. It is to an American author, MacCallum, that the honour is due of having demonstrated this exactly. While studying a hæmatozoon of the raven which is called halteridium, he observed spheroid bodies of two kinds—the one kind hyaline, the other granulated. He then observed the hyaline bodies change to flagellated bodies, from which the flagellæ quickly separated, and one of them was able to penetrate to the interior of a granulated body. Thus the granulated bodies are the female element, susceptible to being fructified by the flagellæ or male element; fifteen or twenty minutes after the fructification the granulated body emitted a conical prolongation, which became more and more elongated, till a mobile little vermicule (called zygote) was given birth to. Further on it will be seen what becomes of it. The observations of MacCallum have been confirmed by Marchoux for the halteridium of the pigeon, by Koch for the proteosoma of the sparrows, and finally by MacCallum and by Grassi for the hæmatozoon of man.

EVOLUTION OF MALARIA.

Etiology.—The existence of malaria in all marshy regions long ago founded the belief that water was the

vehicle of the parasite, and that it was with drinking water that the germs of the illness invaded the digestive canal, to pass thence into the lymphatics and blood. But soon this plausible theory was found not to be in accordance with facts. Then air was accused of transmitting the affection, hence the name "malaria" (bad air) given by the Italians to paludism. But the authors questioned by what means the parasites could thus invade the blood. They began to search the soil and atmospheric dust for the forms of resistance of the morbid germs, but all in vain. Nevertheless this theory had many partisans, when suddenly the question entered other regions, and made rapid progress.

In 1884, Dr. Patrick Manson, formerly a physician in China, and at the present time Director of the London Tropical School, demonstrated the part played by the mosquito in the transmission of another hæmatozoon—the filaria—of the blood. The importance of this fact could not escape Laveran, who then propounded the hypothesis that mosquitoes might also be capable of playing a part in the propagation of malaria, as in that of filariasis.

This hypothesis was at once taken up by Koch, Manson, Ross, Bignami, Dionisi, and finally by Grassi.

Since 1894, P. Manson gave as his opinion that the infection of the mosquito is accomplished by the intermediation of the flagellated bodies, which Laveran with remarkable energy and intuition considered as the most advanced stage of the parasite, while most of the authors still looked upon these as forms of degeneracy. Under the direction of P. Manson, Major Ronald Ross, I.M.S., commenced in 1895 a series of investigations on the propagation of malaria. He tried first to discover directly the cycle of the malaria parasite of man, but his results were quite negative. It was not till 1898 that he conceived the idea of working on birds subject to the *proteosoma* of Labbé, which are hæmatozoa, nearly akin to those of Laveran. He caused birds attacked by malaria to be bitten by mosquitoes, and he observed the evolution of the parasite in the digestive canal of the mosquito; having then caused these infected mosquitoes to bite birds, the blood of which certainly contained no hæmatozoa, he succeeded in transmitting the infection. The cycle of the parasite in the mosquito as observed by Ross will be presently gone into. As the disease of man and that of the bird are analogous, Ross with justice deduced the etiology of one by means of the other. At the end of July, 1898, P. Manson elucidated these facts at the meeting of the British Medical Association at Edinburgh, where the author of this work was present. These important results naturally created a great impression.

Grassi, since 1896, had been conducting investigations on the same subject. He had travelled through all the malarial districts of the peninsula and had arrived at the conclusion that the propagator of malaria in Italy was a particular species of mosquito known by the name of *Anopheles claviger*. He based his statements on the constant presence of these insects in all the infected localities, and on its particular frequency coinciding exactly with that period when cases of malaria are most numerous. En-

couraged by the results obtained by Ross, Grassi associated himself with his colleague Bignami (who at Rome had a hospital ward at his disposal) and with Bastianelli, with whom he proposed to study the fate of the parasite in the body of the mosquito. Grassi collected mosquitoes from an infected locality and took them to a room on an elevated storey of the Hôpital du Saint-Esprit, at Rome. On October 20, 1898, he set a certain number of these *anopheles* at liberty in this room where two persons slept who had spontaneously placed themselves at the doctor's disposal for this experiment. On November 1, the first case of experimental malarial infection made its appearance on one of them. On December 22 of the same year Grassi published the entire cycle of evolution of the hæmatozoa of man in the body of the mosquito. Since that period numerous experiments have been made by Grassi on man. Amongst others he caused a malarial subject to be bitten by healthy *anopheles*; these became infected, and in their turn three of them, by biting a healthy person, inoculated him with the disease. One might still cherish the belief that the mosquito might suck up the germs of the affection from the water of the morasses where it is born, and then at once inoculate man. Grassi proved the contrary; he sought the larvæ and the chrysalis of the *anopheles* in the most malarial districts and reared them in his laboratory; during three months he allowed healthy persons to be bitten by these newly-born *anopheles*, and never observed the slightest accident. From that time he was convinced that in Italy the *anopheles* species is the only carrier of malaria; it infects itself by biting a sick person, and after an evolution of the parasite in its organism, it inoculates a fresh individual.

It will be seen that within a period of a few years the etiology of malaria had made considerable progress. During this time Koch was employed by the German Government to go to the tropics and also to Italy to study malaria. Koch was one of the first to deny the rôle of the mosquitoes; he caused the same to be much discussed by political papers of all countries, but in reality he made more noise than necessary and brought forward no experience whatever in support of the theory. He discouraged Grassi by his railery and malevolent attacks, but the Italian professor had his revenge in the unanimous enthusiastic acclamations which, in last September, greeted the important communication which he made at Munich to the Congress of German Doctors and Naturalists, when he gave a *résumé* of his works on malaria. The author of this article was also present, saw all Grassi's preparations, and is happy to be able to bear testimony that his proofs were most convincing. It is now universally acknowledged that the mosquito is really the propagator of malaria, a fact that Laveran suspected long ago, and which he is pleased to find again confirmed by the works of the eminent Italian.

Evolutionary Cycle of the Parasite.—Two cycles may be observed in the life of the parasite—an endogenous cycle passed in the blood of man, and an exogenous cycle passed in the mosquito.

(1) *Endogenous Cycle.*—The *spheroid bodies* free in the blood may be considered as representing the adult form. The *rosetted bodies* represent the form of

asexual reproduction. These are the agents of auto-infection, *i.e.*, of the dissemination into the organism. As to the crescent forms, these, in the opinion of some authors, represent the encysted forms, that is to say, of the dissemination outside.

(2) *Exogenous Cycle*.—Spheroid bodies and crescent bodies may be eventually absorbed by a mosquito which has just bitten a malarial subject, and the process which we have seen produced in the open air on a microscopic slide is produced in the same fashion, but more quickly, in the digestive tract of the insect. The crescent bodies are transformed into spheroid bodies, and join those that have already been absorbed. Some of these spheroid forms remain intact, being the *macrogametes* or female elements; the others are transformed into flagellated bodies, and the flagellæ separate in order to constitute the microgametes or male elements. The fructification is produced in the stomach of the mosquito, and the fructified gamete becomes a zygote which penetrates into the coats of the stomach, probably forcing itself between the epithelial cells. In this position, highly favourable to its nutrition, the parasite rapidly grows, becomes round, encysted, and from 6 μ finally attains a size of 60 to 80 μ in diameter. At the end of from eight to fifteen days, according to the season, a series of spheres are buried in the cavity of the body of the insect. The interior pigment disappears little by little, and the contents divide into innumerable fusiform sporozoa (about 10,000). At a certain time the capsule ruptures and all the sporozoa become free and spread all over the interior of the mosquito. Then, by a process which is still unknown, they re-assemble in the salivary glands of their host just as a flock wanders in the country to meet again to go back to the stable. The orifices of these salivary glands are situated at the extremity (epipharynx) of one of those lances which are used by the insect to pierce the skin.

The mosquito, in order to prevent the blood from coagulating, inoculates a certain quantity of saliva round the bite, and at the same time it inoculates a certain number of these sporozoa into the blood; these invade the red blood corpuscles and recommence a new asexual generation, as related previously.

It may be that this history of the hæmatozoon may be taxed as a romance, but it is a romance which relates actual facts easy to verify.

To resume, man infects the mosquito, which in its turn infects man. The parasite thus has two residences: one at a constant and elevated temperature in the body of the man, the other at a variable and less high temperature in the body of the mosquito. In other words, the parasite has two alternate hosts, the man and the mosquito.

The Mosquitoes of Malaria.—The mosquitoes belong to that order of insects provided with two wings. Those which seem to play the chief part in the transmission of malaria belong to the family of the Culicidae, or vulgarly speaking, cousins. This family include the species *Culex*, which constitutes the largest group of the cousins, but seems inoffensive, and the species *Anopheles* which seems to be the vehicle of malaria. It is very easy to distinguish one from the other; in the *anopheles* the proboscis is accompanied by two appendices (or palpes) nearly as long as itself,

in the *culex* these palpes are, on the contrary, very short. At least that is what is observed on the females, which alone feed on human blood. The males of both kinds never bite human beings, and can therefore be put on one side; they may be recognised easily by their feathery antennæ. Thus if you are bitten by a mosquito, the proboscis of which seems three-pronged, you have to do with an *anophele*, that is, an insect capable of transmitting malaria to you. Grassi has demonstrated that in Italy the real agent of infection is the *anopheles claviger* (Fabricius). It is characterised by four spots on its wings forming a sort of letter T. Active investigations are being carried on in all countries, and it is probable that before long we shall know the species for each country that is to be blamed.

The *anopheles* are in the habit of biting at sunset, that is to say, at the hour that people linger at their doors to chat or dine. Those insects which do not succeed at this hour take advantage of the night to gorge themselves with blood. This now explains the reason that malaria is above all contracted in the evening, and why it is considered dangerous to pass the night in the open in infected countries. Mosquitoes have a way of dispersing, following a horizontal direction, and only rising a few yards above the soil. That is why elevated localities and the higher storeys of houses are most salubrious.

But the important point is that mosquitoes can only develop in standing water, particularly during the summer months, and that explains why malaria reigns above all during that season, and also in marshy regions. Finally, one can recognise malarial regions by one single examination of the larvæ of mosquitoes in the water. These larvæ are met with in numbers in stagnant waters and water barrels; they float on the surface of the water, head downwards, and at the least stir they flee from all parts, turning many somersaults. The larvæ of the *culex* have at their posterior extremity a peculiar tube which is held above the water to permit them to breathe. The larvæ of the *anopheles*, on the contrary, are not bifurcated at the posterior end, but the respiratory orifices open on the dorsal surface situated at the top of the water. The presence of these latter would naturally give a bad name to the localities where they are met.

TREATMENT.

In order to cause the extinction of malaria two means are open to us: (1) the destruction of the *anopheles*; (2) the cure of malarial subjects.

The first procedure is evidently very difficult, nevertheless in Italy it is being energetically done. Mosquitoes should be relentlessly hunted and destroyed in every corner of habitations where they are met with. One should also strive to kill their larvæ in water, be it by the use of pulverisations of petroleum, be it by aniline dyes. Persons living in malarial districts should avoid going out in the evening or of going to sleep outside after sunset. Windows may be left open if covered by wire netting to prevent the entrance of *anopheles*. It is well to add that by such a proceeding Grassi was able to keep unaffected an entire family in one of the most infested parts of Italy.

But it is evident that the best means will consist in curing all the malarial persons in infected countries. In quinine we have an infallible remedy, and government should make the treatment of malaria compulsory. In reality a malarial person is a danger, not alone to himself, but to every one around him; on the same day he might easily be bitten by twenty anopheles, and these might inoculate hundreds of persons. Thus the best means to destroy malaria is to cure those suffering with the illness. It is not possible to eradicate the disease from the face of the earth, but it would be an appreciable result if it could be exterminated in civilised countries.

Experience demonstrates that quinine should be administered some hours before the attack; it is not always successful in combating the following attack. But it will at least react with certainty on subsequent attacks for a period of five days. But as long as the parasite has not disappeared from the body the illness will recur; thus to avoid relapses, quinine should be ordered in a one gramme dose every five days during a month or six weeks. One should employ the same means as a prophylaxis in infected countries, and thanks to this simple medication one can with impunity go through malarial countries. Malaria is a common, and may become a very serious affection; but on the whole, thanks to quinine, it can no longer be said to be a dangerous affection.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By GEORGE H. F. NUTTALL, M.D., Ph.D.
Pathological Laboratory, Cambridge.

(Continued from p. 277.)

III.

As we have already seen, Manson advanced the hypothesis that the human malarial parasites underwent developmental changes within the body of the mosquito. With the death of the insect, which occurred either in the pools in which it laid its eggs, or on the soil, the parasites became free. Infection was supposed to take place in man by his drinking the water or breathing in the air containing the parasites; in the latter case the micro-organisms being contained in the dust arising from dried pools or the infected soil. Bignami's theory was different. He believed that the mosquitoes acquired the parasite from the infected soil and communicated it to man in the act of sucking blood. Great credit is undoubtedly due to Manson for having stimulated Ross to investigate the matter experimentally. Bignami's hypothesis, on the other hand, also exerted an influence, especially on the direction which experimental research took in Italy. The results of these researches have proved that both Manson and Bignami were partly right in their theoretical conceptions.

DEVELOPMENT OF PROTEOSOMA.

(Takes place in *Culex fatigans* and *Culex nemorosus*.)

Ross (1898, III.) working in Calcutta at a season unsuited for the satisfactory study of the subject on

human malaria, turned his attention to the study of *Halteridium*, and especially the *Proteosoma* (Labbé) infection occurring in sparrows, larks and crows.¹ He observed that—

"(1) Pigmented cells are found in the stomach wall of grey mosquitoes fed on crows, larks and sparrows with *Proteosoma*.

"(2) Pigmented cells are not found in control grey mosquitoes fed on healthy men, or men with crescent plasmodia, or healthy sparrows, on crows and larks, or on crows and pigeons with *Halteridium*.

"(3) These pigmented cells are found in the external coat of the stomach, and grow from a size of 6 μ in thirty hours to 60 μ in six days, and are probably coccidia.

"(4) Successive feeds by the same mosquito on the same bird are followed by fresh crops of young coccidia.

"(5) Similar pigmented cells have been found in mosquitoes fed on human gymnosporidia (Labbé)."

Ross took thirty grey mosquitoes from a batch of grubs procured at one time from the same puddle. Ten (a) of these mosquitoes were fed on a sparrow in whose blood the *Proteosoma* were exceedingly plentiful; ten (b) he fed on a sparrow in whose blood *Proteosoma* was only moderately abundant; and ten (c) he fed on a sparrow whose blood contained no *Proteosoma*. He killed the insects after fifty hours and made counts of the number of pigmented cells in the stomach wall of each mosquito. These countings were repeated by Manson on the same specimens. They found on an average in ten mosquito stomachs belonging to group (a) 100.8 (Ross), and 108.4 (Manson) pigmented cells. In group (b) 29.2 (Ross), and 57.1 (Manson), whilst in group (c) no pigmented cells could be found. Ross states that he has made the same observation repeatedly. Manson, referring to Ross's observations, of which he gives a preliminary report, writes that these show "that certain phases of the hæmatozoal intra-corpuseular parasite of man and birds, after entering the stomach of special species of mosquito, pass into the tissues of the stomach wall of the insect, rapidly increase in size, and probably towards the termination of the life of the mosquito, sporulate, and leave the capsule, which, by that time, has formed around them."² Whilst in London in the beginning of June, 1898, Dr. Manson very kindly showed me Ross's preparations, and I must say that they seemed to me more than suggestive. Laveran,

¹ Koch (1898, I.), as stated previously, expressed himself strongly in favour of the mosquito-malaria theory. Strange to say, he made no mention whatever of the publications of Ross and Manson, though he wrote that he considered experimental work on the mosquito theory to be of extraordinary importance. He said, "Experience has shown that dwellings and sleeping apartments which allow a free passage of air are less to be feared than those in which the air stagnates. It is my conviction that this is due to the latter being preferred by mosquitoes." He, for this reason, recommended the British-Indian bungalow as a model for houses to be erected in the German colonies in Africa.

² Manson gives figures of Ross's preparations showing the mosquito's stomach thirty hours after feeding with *Proteosoma* blood, the pigmented cells lying between the somewhat dis-associated muscle fibres of the stomach wall. Other figures illustrate the development of the parasites in the mosquito, and a diagram is given, indicating what is known and "Yet to be ascertained of the mosquito phase of certain human and avian plasmodia."

who also examined specimens sent to him by Ross, wrote (June 12th, 1898) to Manson, "It appears to me undoubted that the elements discovered by Dr. R. Ross in the stomach of mosquitoes fed on the blood of birds subjects of hæmosporidiosis, are really parasites, and that these parasites represent one of the phases of the evolution of the hæmatozoa. It is probable that it will now be easy to find the extra-corporeal form (*la forme de résistance*) of the parasites in external media. The discovery of Dr. Ross appears to me, as to you, to be of great importance; it is a great step forward in the study of the evolution of the hæmatozoön of birds and very probably also in that of the hæmatozoön of malaria. I have shown the preparations to M. Metschnikoff, who shares my opinion." (Already in another paper Manson and also Lewis are stated to have observed that mosquitoes suck the blood of birds, and that they may perhaps communicate "malaria" to them.)

In Ross's (1898, III.) report published in Calcutta (dated May 21, 1898) full details of his painstaking experiments will be found.¹ Of 245 "gray mosquitoes" fed on birds, with *Proteosoma* 178 (72 per cent.) subsequently contained pigmented cells. Of 249 "gray mosquitoes" fed on men with crescents or immature tertian parasites, on birds with *Halteridium*, on healthy sparrows, on birds with immature *Proteosoma*, "not one contained a single pigmented cell." Of 81 mosquitoes fed on birds known to contain ripe *Proteosoma* 76 (94 per cent.) showed pigmented cells. Ross writes (p. 6), "There can be no question, then, that the pigmented cells are derived directly from the *Proteosoma*. We can, however, already go further. The fact that similar cells were not found in control insects of the same species fed on blood containing other gymnosporidia will convince any one acquainted with parasitology that we are not dealing here with any mere physiological absorption of pigment by the stomach cells of the mosquito, but with a vital phenomenon in the life-history of *Proteosoma*, with a remarkable transformation by which the pigmented parasite in the blood of the bird becomes a pigmented parasite of some kind in the stomach tissues of the mosquito." When the mosquitoes were repeatedly fed on *proteosoma*-blood, a fresh generation of pigmented cells was observed, the parasites of the last feeding being smaller than those at first taken up. The parasites could be seen projecting from the outer surface of the stomach "like warts on a finger." Whereas the earlier forms of the parasites are pigmented, this is no longer the case in the fully-developed individuals.² Where the parasites

are situated between the muscle fibres of the mosquito stomach Ross observes that they remind one of the *Trichina*, from the fact that they cause a displacement of the muscle fibres.

In a letter dated September 7, 1898, Dr. Ross gave me further particulars regarding researches then unpublished. He states that in July he had succeeded in producing *proteosoma*-infection in sparrows, weaver-birds and crows by means of infected mosquitoes. I obtained additional information from Dr. Manson, who had likewise received a communication from Ross, the contents of which he reported at a meeting of the British Association in Edinburgh. (Manson, 1898.) Since the publications already referred to appeared, Ross found that if he crushed the encapsuled *Proteosoma* taken from the mosquito's stomach and placed it in salt solution, that an enormous number of minute spindle-shaped slightly flattened bodies, which he calls "germinal rods," issued from within the capsule. These bodies do not seem to be motile, but they gain access to the body cavity of the insect, and are distributed in all directions by the blood current, and finally accumulate in vast numbers about the 5th to 6th day after feeding *within the cells of the salivary gland*, the cells of which, crammed with the parasites, remind Ross of bacilli-filled lepra-cells. The encysted parasites either give rise to these spindles or to a few "black spores," the significance of which Ross is not prepared to explain.¹ They are not altered if kept for weeks in a moist chamber and do not develop when fed to mosquito grubs. Ross believed from this time that infection results from the bites of mosquitoes whose salivary glands contain the spindle-shaped bodies. Of 28 sparrows 22 acquired severe *proteosoma*-infection as the result of being bitten by infected mosquitoes. Of 4 weaver-birds and 2 crows bitten by infected mosquitoes, only a crow remained healthy. Particularly severe infection resulted in 5 sparrows similarly treated, which had previously been affected by a mild infection. The "gray mosquitoes" used for these experiments in India are found in ditches, puddles, &c., up to an elevation of 7,000 feet above sea-level, and this corresponds according to Ross to the distribution of the *proteosomal* disease in birds.

In a letter dated October 31, 1898, from India, Dr. Ross wrote to me as follows: "The length of time between infecting a mosquito and its being able to cause infection is presumably about seven or eight days, namely, the period it takes the coccidia to mature and the germinal rods to enter the salivary glands." Birds become infected from five to six days after being bitten. The number of mosquitoes required to cause infection has still to be determined. The sparrows used for his mosquito-infection experiments were kept for some days under observation after they had been caught. About 13.5 per cent. of these (111 Calcutta sparrows) were found to be infected. The healthy ones that remained were divided into two lots. "Both lots were kept at night in their cages in separate mosquito-nets. To one lot infected mosquitoes were introduced, while the other (control lot) were preserved from the bites of the mosquitoes

¹ Ross placed the malarial subjects or birds (in cages) he wished to experiment on beneath a mosquito net, and liberated the mosquitoes into the space. After they had filled themselves, the mosquitoes were carefully caught by an assistant entering the net and placing a test tube over each insect. The tube was then closed by a light cotton plug. A few drops of water were placed in the bottom of the tube for the insect to drink and lay her eggs in. The mosquitoes to be kept alive had to be fed on blood every two days. The tubes were changed daily.

² Grassi, Bignami and Bastianelli find that the pigment persists even in the largest capsules formed by human parasites in *Anopheles*. The fact that the pigment is scattered within the much enlarged parasite makes it seem upon superficial examination as if the pigment had disappeared.

¹ The significance of these bodies remains still to be determined (July 2, 1900).

n the laboratory." About 80 per cent. of the sparrows exposed to infected mosquitoes subsequently exhibited *Proteosoma* in their blood, while out of a large number (about forty) of control sparrows only one showed infection on a subsequent examination. In this one sparrow very few parasites were present, and Ross believes that they were simply overlooked at the first examination. After some weeks had passed these control sparrows were in turn exposed to the bites of infected mosquitoes and most of them acquired the disease. As already stated, only fifteen of the 111 sparrows caught exhibited the *Proteosoma* in their blood, but only two of the birds showed more than one parasite in a field. On the other hand, the birds exposed to the mosquito inoculations exhibited an enormous number of parasites in their blood. Ross finds that birds which once show *Proteosoma* always continue to show them in roughly the same numbers. He says it is easy to distinguish between a new and an old infection by the almost invariable presence of the larger, discrete pigment containing parasites, these not being found at the onset of an infection caused by mosquito-bites.

A brief report was subsequently published (February 2, 1899) by the German Malaria Commission, which was composed of Koch, Pfeiffer, and Kossel. The Commission had been in Italy from August 11 to October 2. Koch and his collaborators were able to confirm the work done by Ross, "having come across the right kind of mosquito, which sucks the blood of birds, and in whose stomachs the further development of *Proteosoma* takes place." It is impossible to gather from this statement what species of bird or mosquito was used. The report goes on to say: "We were further able to fill a blank which had been left by Ross, it being proved that after fertilisation had taken place inside the mosquito's stomach, the *Proteosoma* become converted into wormlike bodies, a process which we had already previously discovered with regard to *Halteridium*, a parasite also belonging to this group." After the return of the Commission to Berlin, where the investigations were pursued, the parasites were also found to appear in the salivary glands of the insects. As regards the blank ("Lücke") in the work of Ross to which the Report refers, let me draw attention to the following passage from Ross's "Report on the cultivation of *Proteosoma* Labbé, in grey mosquitoes" (p. 17), which appeared May 21, 1898. Ross says, "Directly I read MacCallum's paper, I found vermicules at once in the stomach of a mosquito killed within an hour of feeding on a crow with *Halteridium*." . . . "The entry of an entire vermicule of *Proteosoma* into the external coat of the stomach of a grey mosquito, and its development there into a pigmented *Coccidium*, afford indeed an explanation fascinating in its simplicity." The Commission reports further: "Noch in einer anderen Richtung ist es uns gelungen, gegenüber den bisherigen Kenntnissen einen Schritt weiter zu kommen. Man nahm bisher allgemein an, dass die sogenannten Halbmondformen und die aus diesen hervorgehenden Geisselkörper degenerirte dem Untergang geweihte Zustände der Malariaparasiten darstellen, hauptsächlich aus dem Grunde, weil sie keine Chromatinfärbung annehmen, ein an und für sich sicheres Zeichen dafür,

dass die Fortpflanzungsfähigkeit solcher Organismen erloschen ist. Durch Verbesserung des hier in Betracht kommenden Romanowsky'schen Färbungsverfahrens konnten wir nun aber Chromatinkörper in den halbmondförmigen Malariaparasiten nachweisen und namentlich auch zeigen, dass die sogenannten Geisseln direkt aus den Chromatinkörper hervorgehen, selbst aus Chromatin bestehen und in Wirklichkeit nicht Geisseln, sondern nach Analogie verwandter Parasiten Spermatozoen sind. In der Verfolgung der Entwicklungsgeschichte des Malariaparasiten weiter vorzudringen ist leider nicht gelungen, aber wir hatten das Glück, einen den menschlichen Malariaparasiten sehr ähnlichen Parasiten bei Vögeln aufzufinden, welcher zur experimentellen Untersuchung sehr geeignet ist. Es ist dies das *Proteosoma*, derselbe Parasit, für welchen der Englische Militär-Arzt Ross in neuerer Zeit den Entwicklungsgang vollständig nachgewiesen haben will."

This portion of the report is decidedly open to criticism. To begin with several investigators have long maintained that the flagellated forms of malarial parasites are not degenerative. It will suffice if I quote in this connection the names of Laveran, Mannaberg, Manson and Dock. It is also well known that Sacharoff (1893 and 1895) found chromatic substance in the flagella of the parasites of birds. He stained the flagella for this purpose by the Romanowsky method, and gives very clear coloured figures and microphotographs of them in both his publications. It would also appear from the report of the Commission as if the process of fertilisation were a new discovery. Everyone familiar with the literature knows, however, that this discovery was made by MacCallum in Baltimore. The remarkable work done by the Italian investigators also receives no mention. In conclusion, Koch makes the following amazing statement: "Die Italiener untersuchen nämlich das Blut im flüssigen Zustande und ohne weitere Hilfsmittel, während ich das Blut im Deckglase antrocknen lasse und, nachdem es fixirt ist, mit Farbstoff behandle. . . ." I do not think further comment is necessary.

(To be continued.)

A HOSPITAL FOR TROPICAL DISEASES IN LIVERPOOL.—A hospital devoted to the treatment of tropical diseases is about to be founded in Liverpool. The hospital is to be erected in memory of the late Miss Mary Kingsley. Many handsome contributions towards the fund have been promised; amongst others Mr. A. L. Jones has given £1,000 and Mr. Blaize £500. The creation of a hospital for tropical diseases in Liverpool marks a new era in medical education in this country. The Seamen's Hospital Society's Branch at the Albert Docks is largely devoted to this purpose, but it shows that the subject of tropical medicine is attaining a still greater hold on the country when a hospital is to be specially built and devoted to this important department of medicine.

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THE

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JULY, 1900.

TYPHOID FEVER AND THE TREATMENT OF THE SICK IN SOUTH AFRICA.

WE are glad to see that the Government immediately grasped the gravity of the statements made by Mr. Burdett-Coutts with reference to the inadequate arrangements in Bloemfontein for the treatment of the sick, and have appointed a commission of enquiry. The selection of the President of the College of Physicians and Professor Cunningham, of Belfast, as the medical commissioners is one which will meet with general approval and inspire confidence that all sides of the question will receive careful attention. Possibly it will be found that the conditions described by Mr. Burdett-Coutts were merely temporary or due to an overflow of the hospitals, owing to an unexpected and unprecedentedly

large outbreak of typhoid fever, and in no way applies to the general system of the hospitals. We are inclined to think so from the letters received from medical men at the front. We are not of those who think that Mr. Burdett-Coutts should have not published these matters. We think he deserves the thanks of the Nation and of the Army in particular. There is nothing in his statements which reflects on the medical department of the Army. Every credit is given it for the extraordinary amount of work which its medical officers performed. Nor do we see that there is any reflection on the Commander-in-Chief. Lord Roberts has to make use of the material with which he is supplied, and if he has, as reported by Dr. Conan Doyle, only a sufficient number of medical men for two army corps, when no fewer than 200,000 men are at the seat of war, it is impossible for him to make them into the requisite numbers. We suspect, if there are any defects, they lie in a system which has for many years rendered the army medical service unpopular and, in consequence, too much depleted, so that even the large additions to the medical department that have been made by the employment of civil surgeons have not been able to bring up the numbers to the requirements of an army 200,000 strong.

Mr. Wyndham gave in his speech some very interesting figures in regard to the mortality from typhoid in the present and in other campaigns. It appears that during this war, and more especially at Bloemfontein, the mortality has been 21 per cent. of the admissions from enteric. In the Nile Campaign of 1898 the mortality from enteric was 28 per cent. In the Dongola Campaign of 1896 it was 50 per cent. ; in the Matabele war of 1896-97, 32 per cent. ; in the Chitral campaign of 1895, 28 per cent. ; in the Soudan Campaign of 1884-85, 39 per cent. This would seem to indicate that either the large number of anti-typhoid inoculations have had a distinct effect on the mortality, or that the treatment the soldiers received was responsible for a lower mortality than usual, or that the virus in South Africa is less fatal. We have always understood that the virulence of the disease is considerable in South Africa, and it will

be interesting and instructive to compare the mortality of typhoid fever among the civil population there in ordinary times and during the war with that which has occurred among the military. Lord Roberts believes that the mortality would be still lower if many of the slight cases diagnosed continued fever, but which were more likely to be mild cases of typhoid, had been included. We have no doubt this contention is perfectly correct. There is not sufficient cognizance taken of the mild cases, either from a statistical point of view or from an epidemiological aspect. We observe that, in the recent report made by the medical commission appointed to investigate the causes of the prevalence of typhoid fever in the American army in Cuba, the mortality only amounted to 7.5 per cent., a figure which appears to have been obtained by including a large number of mild cases.

Whether it is a legitimate classification to place all these cases under the heading of typhoid fever is another question. There is, perhaps, a tendency to go to the extreme and call every disease enteric which has any resemblance to typhoid fever, just as formerly in India every fever was continued or remittent and typhoid was not countenanced. It is not every fever which has ulceration of the bowels that is typhoid. We have been struck by the extraordinary number of deaths from enteric compared with other causes. Is it not possible that many of these enterics are not typhoids? Inquiry in this direction might be most useful in its results. We are sorry that the treatment of the sick has come up in a form which entirely overshadows the far more important question as to the causes of the immense amount of sickness.

BOOTS AND PLAGUE PROPHYLAXIS.—The coolies in Japan, who go bare-footed, are believed to become inoculated by plague through scratches on the feet. On the authorities supplying them with boots plague disappeared from amongst them.

THE Conference on Malaria, proposed to be held in Liverpool during the last week of July, has been postponed. We hope this does not mean that it is to be abandoned altogether.

THE MALARIA MOSQUITO IN EAST AFRICA.

By Dr. SPURRIER.

In the January and February numbers for this year of *The Gazette* for Zanzibar and East Africa, Dr. Spurrier's work in this connection is reported as follows:

A discovery of some considerable importance as regards the health of Zanzibar Island has been made by Dr. Spurrier during the week past. He has found in three places on the outskirts of Zanzibar city the breeding places of the true fever-bearing mosquito, the *Anopheles* of Major Ross. From the larvæ obtained from the pools in question he has succeeded in hatching out in captivity, specimens of the *Anopheles* mosquito.

What is perhaps also of great importance is that he has likewise found a larva which preys upon the mosquito larvæ to a most formidable extent, one of such on two occasions devouring twelve victims within a few hours. These larvæ are also being hatched in captivity and develop into an insect similar to a flying ant, with two long delicate tail-like appendages.

The importance of finding the breeding places of the fever mosquito is very great as they are easily overlooked. In India, Major Ross had in many localities to search several square miles before the larvæ were found. There are three conditions necessary and must be co-existing.

(1) The breeding pools must be large enough to be permanent for a few days to allow the larvæ to mature.

(2) They must not be so large as to contain minnows, frogs, and such like, which devour larvæ with avidity.

(3) They must not be liable to be scoured out by every shower of rain.

The larvæ are found floating parallel to the surface of the water, differing from the common or *Culex* mosquito larvæ which float head downwards.

On the inner margin of each main wing of the *Anopheles* are four black spots visible to the naked eye, the proboscis is very prominent and is prolonged in the same line as the chest and head; the body of the insect projects from any resting surface boldly, due to the great length of the two hind legs. It is a very business-like looking insect, and once seen cannot to the least observant person be confused with other species of the mosquito.

The matter of the "malaria mosquito" is still occupying Dr. Spurrier's attention, and in a recent visit to Dunga, his research was rewarded by the discovery of the existence of this peccant insect in that district also.

Major Ross claims that by obliterating breeding places of this insect the whole character of a fever district may be altered.

To be able to turn the suburbs of Zanzibar into healthy residential sites should be sufficient inducement to pursue this subject.

The districts found to possess the breeding places, are Kiungani, Migombani, and the top swamp of "Holmwood's shamba."

Owners of shambas have it within their power by taking concerted action to free their own districts from this harmful insect. The breeding places are very few in number and once found may be easily obliterated by drainage or treated by Major Ross's kerosine method. In the Zanzibar district which has up to now been thoroughly examined, only three breeding pools were found in a square mile of ground. The mosquito of fever continues to flourish not by the difficulties of dealing with his location, so much as the harmful part he plays in the production of disease failing to convince the mind of residents in fever districts, and the new teaching being looked upon as mere babble.

The larvæ of the fever-bearing Mosquito *Anopheles* have now been found at Dunga in the puddles, formed by the spilling of water at the small dip holes made in the drying swamps, on the Zanzibar side of the Dungan eminence.

The evidence for the concomitance of the Fever Mosquito with admittedly feverish localities is thus growing. The burden of proof for the causation is still considered by many to rest on the shoulders of the disciples of the Mosquito theory.

A spot map of Zanzibar and its environs is now being made showing mosquito-infected localities, and the species of mosquito up till now found therein. The co-operation of the interested public in this important work is earnestly solicited.

The Malaria Mosquito.—Dr. Spurrier contributes some additional notes on the fever-bearing mosquito—the breeding places of which he has recently found in three notoriously unhealthy districts, Kiungani, Migombani and the top swamp of Holmwood's shamba.

He has now succeeded in finding the larvæ of the mosquito in two new situations (1)—in a disused stone water-tank in the town—contrary to what has been hitherto generally stated, the breeding places having been asserted to be confined to natural puddles in the country; and (2) in the hollows formed between the ridges of earth on which the sweet potato is cultivated; the water collected there he has found to contain abundant larvæ.

The larvæ may not be seen on first inspection of a pool or tank. As they lie parallel to the surface they are liable to be blown to and fro by the wind.

As a protection they cling by their tails to any reeds or grasses in the water, or to the sides of a tank; on touching the grasses at the edge, the larvæ shoot out into the middle and may be dipped up if the hand of the observer is quick enough, for they are very smart in their action.

Too much water is as a rule taken up in catching the larvæ, by tapping the bottle or dipper they at once descend and the superfluous water can be poured off.

More specimens of the "destroyer" larvæ have been obtained, equally voracious to those first caught.

THE PREVALENCE OF PLAGUE.

In India generally plague is subsiding in extent and virulence. The fourth epidemic in Bombay is nearly over, the deaths from plague amounting to less than 100 weekly. In Calcutta what may be termed the first epidemic is subsiding, but the course of the disease in Calcutta differs in its behaviour from almost all other cities. Appearing first in a sporadic form, it seemed to become endemic, and only within the last few months has it shown itself in at all an epidemic form. Again, the plague mortality in Calcutta has relapsed into what seems a normal quantity, and the term "remittent" seems best to express the behaviour of plague in Calcutta. In the Bombay Presidency, at Kurachi, and Hyderabad, and at Aden, a few cases of plague only are met with at present.

From Hong-Kong the plague report is unsatisfactory in the extreme. From 60 to 100 deaths weekly are reported from there, showing a marked increase upon the earlier months of the year—the cold weather season.

It is a matter of grave moment the revival of plague in Hong-Kong at the present moment. Fleets of all nations are assembling there; soldiers of most of the European countries are passing through the port, and Europeans from all the coast ports are hastening thither, some to escape from actual, and many from threatened, dangers. The danger is evident, and how to guard against it is one which concerns the peoples of all nationalities. We are aware that the Hong-Kong sanitary authorities are doing their utmost to meet the situation; but in our present state of knowledge in regard to the communicability of plague it would be ungenerous to attempt to fix blame upon them, should the dissemination of plague in the Far East assume alarming proportions.

In Australia plague does not seem to have attained a serious hold, and being less in the highway of the world's commerce than such places as Hong-Kong, Port Said, and Oporto, the limitation of infection is more possible, and sanitary efforts likely to meet with better results than elsewhere.

In Oporto a case of plague was reported on June 20. The fact may not be of great moment, but it proclaims the imminent danger there is of a recurrence of plague in Portugal and its spread elsewhere in Europe. From the fact that the Lisbon residents are leaving for Madeira a month earlier this year than usual, it behoves all concerned to keep an eye upon Oporto and to be well informed as to the actual state of plague there. The people who have gone to Madeira from Lisbon, declare that plague is in the country, and they left Portugal earlier than usual owing to the dread of the imposition of quarantine. This may be a mere scare, but it is not a reassuring statement.

In Rio de Janeiro plague seems to linger; if we can trust the evidence before us it would appear that out of 200 cases of plague which have occurred since the commencement of the outbreak only 11 have died. A $5\frac{1}{2}$ per cent. death rate from plague is an unheard-of result, and we are inclined to question the accuracy of the returns, or even the certainty that it is really plague that exists in South America.

THE DISSEMINATION OF PLAGUE.

THE Egyptian sanitary authorities did good work when they set on foot a minute and searching enquiry into the origin of plague in Alexandria. The report of a special Commission has just been published, and although the results are mostly of a negative character, the one positive result arrived at, that plague was not imported into Egypt from Bombay, is important. It was popularly believed that plague was carried to Egypt by some Gallicians who reached Alexandria from Bombay; this myth, however, has been finally exploded. Attention was therefore directed to the infection of imported goods, and chiefly grain, but here again a negative conclusion was arrived at. Almost all goods coming to Alexandria from Bombay arrive at Port Said, and the fact that Port Said was not affected in 1899 would seem to imply that infection was not carried by that channel.

Experimental evidence goes to show that the power of infection of grain lasts but a few days, and long before the distance between Bombay and Alexandria could be covered plague infection by such a source is impossible.

The existence of plague in Jeddah, the port of Mecca on the Red Sea, in 1898, must be taken into account, as Jeddah is in fairly frequent communication between both Suez and Alexandria. But the Egyptian Sanitary Board especially prides itself upon the careful control of the pilgrim traffic, and the most diligent search fails to connect the Arabian outbreak with the appearance of plague in Alexandria.

As usual with plague epidemics elsewhere, it is quite impossible to determine the exact period when the first case of plague existed. It is now believed that plague cases occurred in Alexandria before May, 1899, and no sooner was the disease officially recognised than retrospective diagnoses became rife, and it is now fairly certain that cases of plague occurred in Alexandria two or three months before it was recognised.

As regards the spread of plague to Australia and the French colony of New Caledonia, no more positive results have been arrived at. The French hold that plague was brought to New Caledonia by gunny bags from Calcutta. All such shipments, however, pass through Sydney, and it would seem that the fact of Sydney and New Caledonia being both affected was conclusive evidence of the connection. But the ships that carried cargo of this kind distributed part of the same cargo to New Zealand, Queensland and various other Australian ports, several of which have been free from plague. The Australian Plague Conference came to no conclusion as to the origin of the disease in Australia. The rat, however, is viewed with suspicion by the Australians as being the most probable source of infection, and they, in common with people of many other nationalities, are devoting their attention in the infected parts to the destruction of the rat.

Reply to Article for Discussion.

CANCER IN INDIA.

WITH reference to your note on prevalence of cancer in the tropics I may make the following remarks. In "Evolution and Disease" Mr. Bland Sutton, if I remember aright, states that cancers are rare in the dark races. Chevers states that cancer "was by no means remarkably prevalent in India," but still every hospital sees cases.

Epithelial cancer is the most prevalent form, I believe, of skin (especially from burns in Kashmir, Neve), conjunctivæ, penis, tongue, and uterus (see Sir J. Fayrer's "Clinical Observations in India").

In the museum of the Calcutta Medical College there are specimens of scirrhus and medullary cancer.

On the whole I do not think cancer or other malignant disease is by any means so common among natives of India as in Europe. Among British and native troops, and among prisoners (for all of whom accurate statistics exist), there are very few admissions to hospital recorded for these diseases.

Yours, &c.,

W. J. BUCHANAN, M.B.,

Major I.M.S.

Simla, June, 1900.

Reprints.

A CUTEREBRAL LARVA IN THE EYE-LID.¹

By Dr. J. BLEYER.

Cidade de Lages, Santa Catharina, Brazil.

(Translated from the German by P. Falcke.)

DISTRIBUTED over the whole of tropical America, and existing principally in Brazil, there are several of the genus *Cuterebra* or *Dermatobia* which, while belonging to the family of European gad-flies, yet differ materially from the latter. These *Cuterebra* have the peculiarity of depositing their ova under the cuticle of human beings and animals.

One species observed by me was distinguished by a broad yellow stripe on its posterior part, with blackish antennæ and reddish-brown faceted eyes; it re-

¹ On the authority of Professor Nuttall, Cambridge, "the genus *Cuterebra* (Clark, 1815), comprised under the family *Cæstridae*, is allied to the genus *Hypoderma*, &c. The *Cuterebra* belong to the *Diptera*."

sembled the common blue-bottle, but was somewhat larger. I repeatedly found the larvæ of this fly under the swollen skin of men and animals, and forming a peculiar inflammation in the shape of a boil. The boil, which contains a single ovum of this species of fly, and in which later the larva at the commencement of its development resembles a faruncular nodule, soon increases in circumference, secreting a lymph-like fluid. One larva only is found in each boil, which when abstracted by means of forceps is, if fully grown, 26 mm. in length and 5 mm. in breadth. It feeds upon the lymphatic secretion which it causes under the tissues of the skin. These boil-like inflammations are found on various parts of the human body, mostly on the surface of the back and on the upper and lower extremities, but also on the scrotum and on the vulvæ, giving rise to a suspicion of local sores.

Some time ago I observed one of these remarkable larvæ in the lower eyelid of a child, a girl aged 3, who was suffering with conjunctivitis. The boil, which was situated on the inner side of the eyelid, had caused a sort of ectropion and was still developing. The tumour, of a reddish colour and of a granular consistency, was about the size of a raspberry. The child's parents suspected that it was a malignant tumour of the eyelid.

The extraction of this fly-larva is accomplished by enlarging the aperture at the apex of the boil from which frequently part of the body of the larva protrudes, and then seizing the larva with forceps.

In the case of sensitive persons, particularly children, the application of a little æthyl-chloride is necessary in order to produce local anæsthesia, before incising the skin.

After removal of the parasite the injection of a little emulsion of creoline (weak solution) is recommended, followed by the application of iodoform-collodion, and a bandage with iodoform gauze; or better still, the insertion into the cavity of the wound of some iodoform-lanoline (R: iodoformii 0.5, lanolini 15.0, olei amygd. dulc. 20.0). These measures are indicated because, occasionally after removal of the cuterebral larva, it is very easy for the dangerous *Lucilia hominivora* laqu. and other species of flies to invade the cavity of such a boil for the purpose of there depositing their ova or their small living larvæ. Such a secondary infection with these new fly-larvæ, even in small numbers (generally 20 or 50 to 300) may lead to serious destruction of the muscles, nerves and vessels, in consequence of these all-devouring larvæ finding their way into the deeper layers, thus causing an agonising death to man and beast.

Cases of this kind frequently take place in the interior of Brazil and have been repeatedly observed by the author. Wounds and ulcerative processes attacked by some species of flies, and which are much more dangerous than the boil originated by the *Cuterebra*, are treated successfully with liquor aluminii acetici and acidum pyrolignosum in a diluted solution.

Amongst the indigenous population of this country such "bicheiros" are treated with tobacco juice and calomel, symptoms of poisoning often resulting. Lately, thanks to the advice of more experienced

persons, particularly the doctors, creolinum (Pearson) and similar preparations are being used, not only for human beings but for animals, who often suffer from the plague of flies.

For the relief of the animals the "tropeiros" (leaders of mule caravans) use the creolin undiluted, introducing the same by means of small feathers into the swelling containing the larvæ. After the expulsion of the parasites the cavity of the wound is closed with clay or fresh dung. The "facondeiros" (land-owners) use the same method in the camp districts to free the cattle of the "fly boils."

As a prophylactic measure it is recommended to the inhabitants of those regions in which these injurious species of fly abound, to exercise minute supervision and great cleanliness over every part of the body which is subject to perspiration. It is advisable to wash the exposed parts of the body with tepid or cold water to which some strong vinegar or eau de cologne has been added. In tropical climates, invalids and children who suffer with conjunctivitis, catarrh of the ears, bleeding of the nose, eczema, ulcers or wounds, should be carefully guarded from the attacks of dangerous flies. Carelessness as regards this rule may often have the saddest results. Finally, from the hygienic point of view, the destruction of all species of flies in dwelling- and sick-rooms is urgently to be pressed, as they have been proved to be disseminators of dangerous infectious forms of disease, such as tuberculosis, anthrax, typhoid, yellow fever and plague. — *Archiv für Schiffs- und Tropen-Hygiene*, May, 1900.

FILARIA NOCTURNA IN CULEX: PROBABLE MODE OF INFECTION OF MAN.

By GEORGE C. LOW, M.A., M.B. Edin.

Craggs Scholar, London School of Tropical Medicine.

THE coloured plate which we are permitted to print this week, through the courtesy of the *British Medical Journal*, illustrates most of what was previously known concerning the disposition of filaria in the body of the mosquito, and also adds new facts indicative of the possibility of the mosquito bite causing infection in man.

In filariated mosquitoes (*Culex ciliaris*), sent to Dr. Manson from Australia by Dr. Bancroft, Dr. Low, at the London School of Tropical Medicine, discovered the filaria passing into the proboscis of the mosquito. The parasite does not pass along the salivary duct, but makes an independent passage through the base of the labium and pushes forwards along the proboscis between the labium and the hypopharynx. It seems fair, therefore, to conclude that when the mosquito bites the filaria finds its way in the human body along the proboscis.

DESCRIPTION OF COLOURED PLATE,

Fig. 1.—*Filaria nocturna* in the blood in the stomach of newly fed mosquito, $\times 60$. Fig. 1 a, the same, isolated, $\times 125$. (Australia.)

Fig. 2.—*F. nocturna* lying in thoracic muscle of mosquito after quitting stomach; first day after

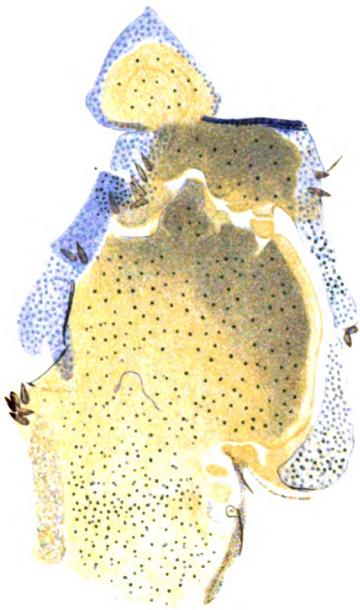


Fig. 1.



Fig. 1a.

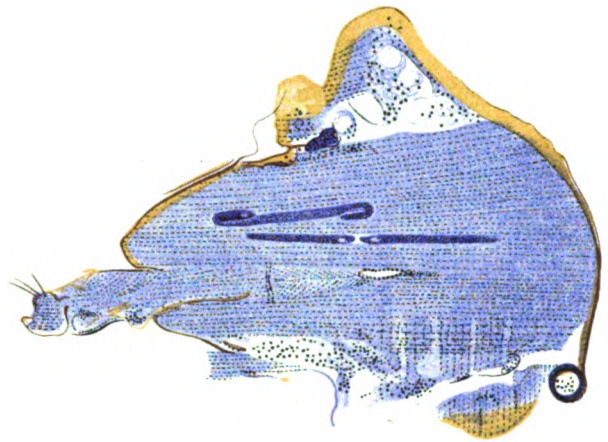


Fig. 4.

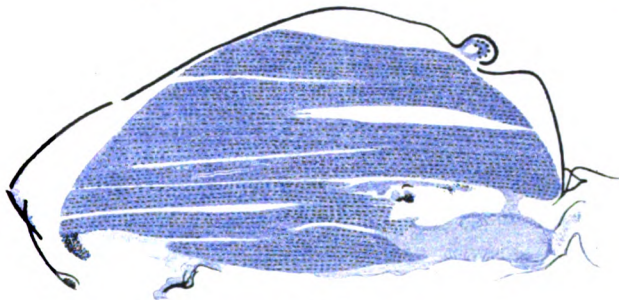


Fig. 2.



Fig. 2a.

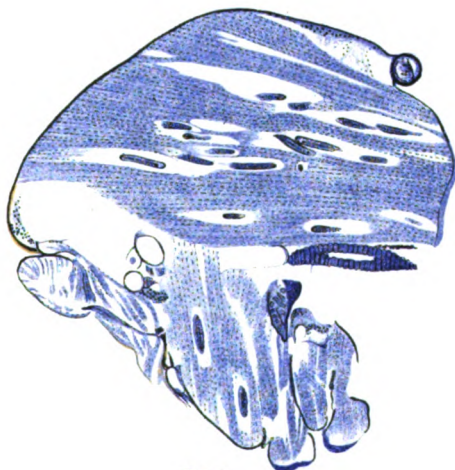


Fig. 3.

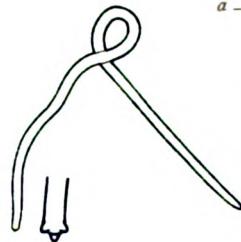


Fig. 7.

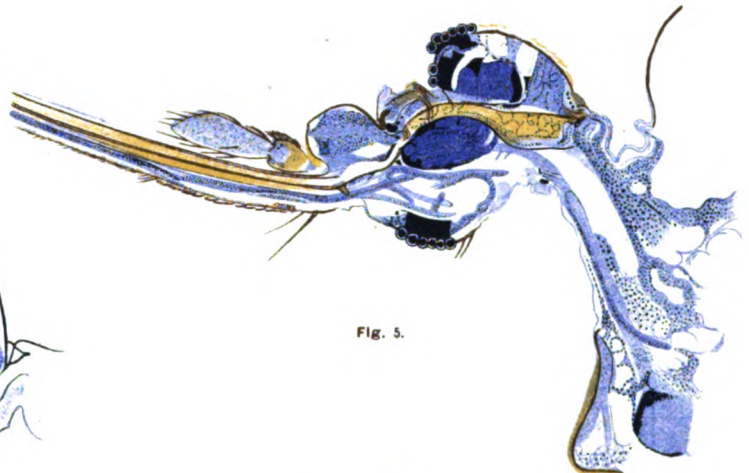


Fig. 5.

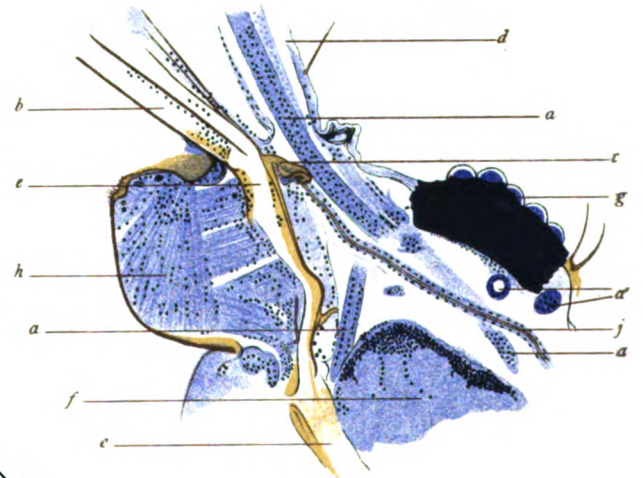


Fig. 6.

EVOLUTION OF *FILARIA NOCTURNA* IN *CULEX*.
Reprinted by permission of the *British Medical Journal*.

- feeding, $\times 60$. Fig. 2 a, the same, isolated, $\times 125$. (China.)
- Fig. 3.—*F. nocturna* in thoracic muscle of mosquito 9 days after feeding. Great increase in size; commencing differentiation of alimentary canal. (Madras.)
- Fig. 4.—*F. nocturna* in thoracic muscles 11½ days after feeding; development nearly complete. (Australia.)
- Fig. 5.—*F. nocturna* in head and proboscis of mosquito 20 days after feeding. Shows parasites coiled up below cephalic ganglion and extending along proboscis between labium and hypopharynx, $\times 60$; part of another filaria can be seen passing along the neck from the prothorax to the head. (Australia.)
- Fig. 6.—High power view of another section showing exact route by which the filaria enters the proboscis. The salivary duct is seen entering base of hypopharynx. (a) Fragments of filaria; (b) labrum; (c) base of hypopharynx; (d) labium; (e) pharynx; (f) cephalic ganglion; (g) eye; (h) muscle; (j) salivary duct opening into base of hypopharynx. (Australia.)
- Fig. 7.—Filaria dissected out of mosquito 50 days after feeding. The tail with its appendages shown more highly magnified. (Australia.)

New Drugs, &c.

LEVURINE.—In the *Scottish Medical and Surgical Journal*, April, 1900, Professor Simpson records several cases of pyrexia treated by levurine with apparent benefit. Levurine is no new drug, but the yeast of beer prepared in a concentrated form and exhibited as a powder with aerated water, beer, or in cachets. The dose is a teaspoonful thrice daily if necessary and given before food. It is well worth a trial in the tropics; so far it has been used in high temperatures in tonsillitis, uterine and pelvic inflammation, carbuncle, acute pneumonia, &c., and in all with apparent success. It would be interesting to have a careful trial of levurine in cases of malarial fevers. The drug may be obtained from Wm. Martindale, 10, New Cavendish Street, Portland Place, London, W.

Special News and Notes.

BRITISH MEDICAL ASSOCIATION.

TROPICAL SECTION.

At the meeting of the Association at Ipswich, on July 31 and following days, the Tropical Section will meet in the ART ROOM, SECOND FLOOR, HIGHER GRADE SCHOOL.

President: Colonel KENNETH MACLEOD, M.D. (ret. I.M.S.). **Vice-Presidents:** Surgeon-Lieutenant-Colonel OSWALD BAKER, M.D., I.M.S.; Major RONALD ROSS, I.M.S. **Honorary Secretaries:** ANDREW DUNCAN, M.D., M.R.C.P., School of Tropical Diseases, Albert Dock, E.; GUTHRIE RANKIN, M.D., M.R.C.P., 4, Chesham Street, Belgrave Square, S.W.

On Wednesday the business will be introduced by a short address from the President, Colonel Kenneth Macleod.

The following subjects have been arranged for discussion:

Wednesday, August 1.—Quinine: its Action and Modes of Employment in Malaria. The discussion on this question will be opened by Dr. Andrew Duncan, M.R.C.P. The following will take part in the discussion: Lieutenant-Colonel Stewart, I.M.S.

Thursday, August 2.—Ankylostomiasis. The discussion on this question will be opened by Major G. M. Giles, M.B., F.R.C.S.

Friday, August 3.—The Etiological and Pathological Relationships of Yaws. The discussion on this question will be opened by Mr. Jonathan Hutchinson, F.R.S., LL.D.

The following gentlemen have intimated their intention to take part in the discussions: Dr. Patrick Manson, Mr. James Cantlie, Colonel Kenneth Macleod, Deputy Inspector-General John Tyndall, Mr. Malcolm Morris, Surgeon-Lieutenant-Colonel Oswald Baker, Sir Wm. Kynsey, Major Wilson, Dr. Fielding-Ould, Dr. Alfred Hillier, Dr. James Watson, Dr. Sandwith (Cairo), Dr. Thin, Major W. J. Buchanan, I.M.S., Major F. P. Maynard, I.M.S., Lieutenant-Colonel Maitland, I.M.S.

The following papers have been proposed:

BASSETT-SMITH, Percy W., Staff Surgeon, R.N. Abscess of Left Lobe of Liver, with Remarks, particularly referring to the Amœbic Causation. BUCHANAN, Major W. J., M.B. Hot Weather Diarrhoea in India.

CANTLIE, James, F.R.C.S. Subhepatic Abscess.

DUNCAN, Andrew, M.D. The Diseases of Ghorkhas. MACLEOD, Colonel Kenneth, M.D. The Management of Lung Lesions consequent on Liver Abscess.

SAMBON, Louis, M.D. The Geographical Distribution and Natural History of Plague.

SMITH, W. Johnson, F.R.C.S. The Surgical Treatment of Hepatic Abscess.

THIN, George, M.D. A note on a case of Blackwater Fever, with Specimens.

WILLIAMSON, George A., M.D. The Cyprus Sphalangi and its Connection with Anthrax.

Members proposing to take part in the discussions or desirous of reading papers should communicate, without delay, with the Honorary Secretaries.

INOCULATION AGAINST TYPHOID.

IMPORTANT statistics have at last been made public concerning the probable effects of inoculation against typhoid. The figures relating to the results have been communicated by Dr. A. E. Wright, professor of pathology in the Army Medical School, Netley, and

they are founded upon the experience gained in Ladysmith during the siege. The number of officers and men under observation numbered 12,234, of these, 1,705 had been inoculated, leaving 10,529 non-inoculated or non-protected. During the siege, 1,489 cases of typhoid fever occurred amongst the non-inoculated and 35 amongst the inoculated. From these figures it is seen that 1 in 7.07 of the non-inoculated contracted typhoid, whilst only 1 in 48.7 of the inoculated were attacked. The lessened mortality also amongst the inoculated is marked; for of the non-inoculated persons attacked 329, or 1 in 32 died; whilst amongst the protected, 8 deaths only occurred, or 1 in 213. Professor Wright remarks that in the returns "the inoculated" are not definitely specified; inoculation being made to include, in all probability, persons treated by either the anti-typhoid serum or the sterilised vaccine culture, or even vaccination against small-pox. We await with interest a full investigation and report on this most important and interesting question, and it is to be hoped that Professor Wright's treatment may be proved to be a real protection.

Letters, Communications, &c., have been received from:—

- C.—Mr. Luiz A. da Costa, V.C., London.
 E.—Miss Wynne Edwards, Denbigh.
 F.—Dr. Fernandes Figueira, Rio de Janeiro.
 G.—Dr. J. G. Gimlette, Barnam.
 H.—Dr. Harrison, London. Mr. C. W. Hewlett, London.
 Major H. Hendley, Lahore. Staff Surgeon V. Hooper, R.N., China.
 L.—Sir Francis Lovell, Port of Spain.
 M.—Mr. H. Mirjakawa, London. Dr. Wm. McDonald, Antigua.
 N.—Dr. G. H. Nuttall, Cambridge.
 P.—Dr. Tottenham Posnett, Kildare.
 R.—Dr. Rho, Rome.
 S.—Dr. Sandilands, New Hebrides. Dr. A. Stanley Shanghai.
 Y.—Major M. T. Yarr, Bloemfontein.

EXCHANGES.

Annali di Medicina Navale.
 Archiv. für Schiffs u. Tropen Hygiene.
 Archives de Medicine Navale.
 Australasian Medical Gazette.
 Boletín de Medicina Naval.
 Boston Medical and Surgical Journal.
 Bristol Medico-Chirurgical Journal.
 British and Colonial Druggist.
 British Journal of Dermatology.
 British Medical Journal.
 Climate.

Clinical Journal.
 Clinical Review.
 Giornale Medico del R. Esercito.
 Il Policlinico.
 Indian Engineering.
 Indian Medical Gazette.
 Indian Medical Record.
 Janus.
 Journal of Balneology and Climatology.
 Journal of Laryngology and Otology.
 La Grèce Médicale.
 Lancet.
 Liverpool Medico-Chirurgical Journal.
 Medical Brief.
 Medical Missionary Journal.
 Medical Record.
 Merck's Archives.
 New York Medical Journal.
 Pacific Medical Journal.
 Polyclinic.
 Public Health.
 Revista Medica de S. Paulo.
 South African Medical Journal.
 The Hospital.
 The Medical and Surgical Review of Reviews.
 The Northumberland and Durham Medical Journal.
 Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."



